A STUDY ON AN ANALYSIS OF THE ECG, CHEST X-RAY, PULMONARY FUNCTION TESTS, PULSE OXIMETRY, HAEMATOCRIT ABNORMALITIES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

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CERTIFICATE

This is to certify that the dissertation entitled **"**AN ANALYSIS OF ECG, CHEST X-RAY, PULMONARY FUNCTION TESTS, PULSE OXIMETRY, and HAEMATOCRIT ABNORMALITIES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS" is the bonafide original work of **Dr. P. Sasikumar** in partial fulfillment of the requirements for M.D. Branch-I (General Medicine) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in April 2012.

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I **Dr. P. Sasikumar**, solemnly declare that dissertation titled, **"**AN ANALYSIS OF ECG, CHEST X-RAY, PULMONARY FUNCTION TESTS, PULSE OXIMETRY, HAEMATOCRIT ABNORMALITIES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS**"** is a bonafide work done by me at K.A.P.V. Government Medical College, during 2009-2012 under the guidance and supervision of my Unit Chief **Prof. Dr. P. KANAGARAJ, M.D.,** Associate Professor of Medicine, Chief – Medical Unit – II.

The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree (Branch – I) in General Medicine.

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CONTENTS

INTRODUCTION

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. Many people suffer from this disease for years and die prematurely from it or its complications. COPD is the fourth leading cause of death in the world[9] and further increases in its prevalence and mortality can be predicted in the coming decades[8].

In the definition of COPD, the phrase "preventable and treatable" has been incorporated following the ATS/ERS recommendations to recognize the need to present a positive outlook for patients, to encourage the health care community to take a more active role in developing programs for COPD prevention and to stimulate effective management programs to treat those with the disease[2,8]

The chronic airflow limitation characteristic of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person.[8] COPD has a variable natural history and not all individuals follow the same course[7]. However, COPD is generally a progressive disease, especially if a patient's exposure to noxious agents continues[1,8]. The impact of COPD on an individual patient depends on the severity of symptoms (especially breathlessness and decreased exercise capacity) systemic effects, and any comorbidities the patient may have not just on the degree of airflow limitation.

Worldwide, cigarette smoking is the most commonly encountered risk factor for COPD, although in many countries, air pollution resulting from the burning of wood and other biomass fuels has also been identified as a COPD risk factor.[8]

The prevalence of COPD reported in different population based studies from India is highly variable[7]. The prevalence rates in male subjects of 2.12% to 9.4% in studies reported from North are generally higher than 1.4% to 4.08% reported from South India. The respective ranges for female subjects vary from 1.33% to 4.9% from North and from 2.55% to 2.7% from South India. For epidemiological assessment, the rounded-off median prevalence rates were assessed as 5 percent for male and 2.7 percent for female subjects of over 30 years of age. The disease is distinctly more common in males. The male to female ratio had varied from 1.32:1 to 2.6:1 in different studies with a median ratio of 1.6:1[7]

Spirometry provides quick assessment of expiratory functions that correlate with FEV1 & also enable us to differentiate between restrictive, obstructive & proximal air way disease[8]. The combination of Pulse oximetry & Spirometry give valuable information about patient's status. Long standing COPD disease can lead to exertional & nocturnal hypoxemia. Frequent Hypoxic episodes & nocturnal hypoxemia leads to the development of secondary polycythemia & its consequences.[10]

AIM OF THE STUDY

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To study age and sex distribution in COPD patients

To study the risk factors in COPD patients.

To study about the duration and influence of smoking habit in the development& progression of COPD.

To study the presenting symptoms in COPD patients

To study the physical signs in COPD patients

To Study the Pulse oximetry values to detect hypoxemia, prognosis, to plan oxygen therapies in chronic obstructive pulmonary disease.

To study the correlation between Hemoglobin level and severity of disease.

To study the correlation between Hematocrit level and severity of disease

To assess the severity of chronic obstructive pulmonary disease

by the pulmonary function tests.

To study chest x-ray findings, ECG findings in COPD patients

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

Arteams (260 AD) had given many clinical descriptions of pulmonary disease of which 'pneumodes' might be the same as chronic bronchitis, emphysema or bronchial asthma leading to congestive cardiac failure.

In Europe, Bodham (1808) and Laennec (1827) made the classic description of chronic bronchitis & emphysema.

Laennec in 1926 described the clinical and pathological characteristics of cause heart failure in otherwise healthy heart.

In 1934 Kountz and Alexander who were studying emphysema stated that "it appears that heart is affected in majority of patients with emphysema".

I.T.T. Higgins in 1959 had studied men between the ages of 25 and 64 to know the relationship between smoking and respiratory symptoms. A clear relationship between smoking and persistent cough and sputum production has been found.

In 1959 The Medical Research Council in its publications used the term chronic bronchitis to define expectoration when other causes such as bronchiectasis or tuberculosis have been excluded, to patients who have coughed up sputum on most days during at least for 3 consecutive months in 2 successive years

1963 Christer Larson studied 246 Swedish adults with severe α1 –antitrypsin deficiency; primary emphysema was present in 109cases.

By 1964, the Advisory Committee to the Surgeon General of the United States had concluded that cigarette smoking was a major risk factor for mortality from chronic bronchitis and emphysema. Subsequent longitudinal studies have shown accelerated decline in the volume of air exhaled within the first second of the forced expiratory maneuver (FEV_1) in a dose-response relationship to the intensity of cigarette smoking, which is typically expressed as pack-years (average number of packs of cigarette smoked per day multiplied by total number of years of smoking)

1975, Earliest nocturnal polygraph studies were done

1977, Flick MR et al, did a land mark study is to show that Patients with COPD experienced a worsening of hypoxia.

1964-1973 Boushy and colleagues published a series of papers on the subject of prognostic factors in COPD and the prognostic values of lung function tests

1977 Boushy SF et al. described the results of hemodynamic changes in 136 patients with COPD including serial studies, correlating pulmonary function tests, arterial oxygen pressure, and arterial carbon dioxide tension with hemodynamic parameters.

1983, Catterall TR et al., studied about transient hypoxemia during sleep in COPD patients. Long-term oxygen in conjunction with pulmonary rehabilitation, it also improves quality of life

1991, Weitzen blume et.al. analysed the evaluation of physiological variables in patients with COPD before & during long term oxygen therapy.

DEFINITION

Chronic obstructive pulmonary disease (COPD) is characterized by chronic airflow limitation and a range of pathological changes in the lung, some significant extra pulmonary effects, and important comorbidities which may contribute to the severity of the disease in individual patients. Thus, COPD should be regarded as a pulmonary disease, but these significant comorbidities must be taken into account in a comprehensive diagnostic assessment of severity and in determining appropriate treatment.[1,7,8]

Based on current knowledge, a working definition is:

Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable disease with some significant extra pulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases.[8]

Airflow Limitation in COPD

The chronic airflow limitation characteristic of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person. Chronic inflammation causes structural changes and narrowing of the small airways. Destruction of the lung parenchyma, also by inflammatory processes, leads

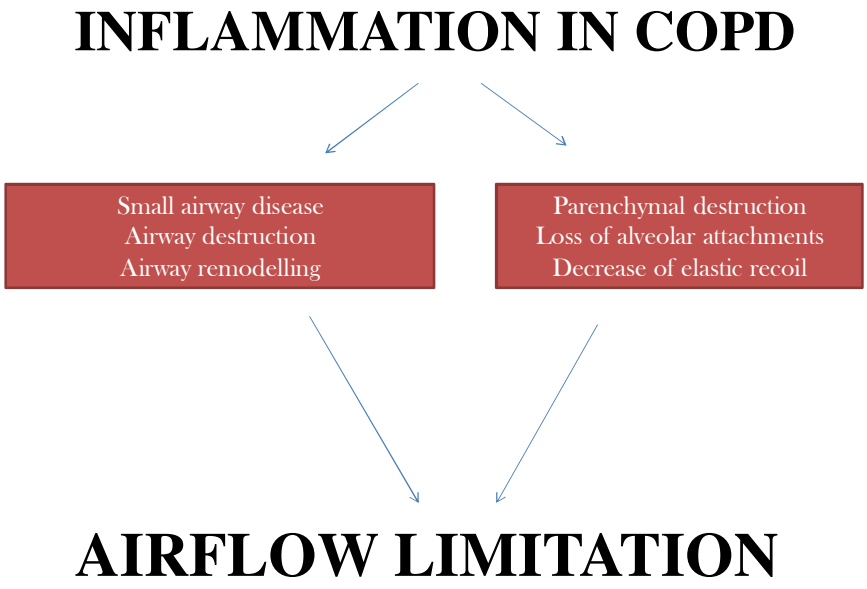


Figure.1

to the loss of alveolar attachments to the small airways and decreases lung elastic recoil; in turn, these changes diminish the ability of the airways to remain open during expiration. Airflow limitation is best measured by Spirometry, as this is the most widely available, reproducible test of lung function Many previous definitions of COPD have emphasized the terms “emphysema” and “chronic bronchitis,” which are not included in the definition used in this and earlier GOLD reports. Emphysema, or destruction of the gas exchanging surfaces of the lung (alveoli), is a pathological term that is often (but incorrectly) used clinically and describes only one of several structural abnormalities present in patients with COPD. Chronic bronchitis, or the presence of cough and sputum production for at least 3 months in each of two consecutive years, remains a clinically and epidemiologically useful term. However, it does not reflect the major impact of airflow limitation on morbidity and mortality in COPD patients. It is also important to recognize that cough and sputum

production may precede the development of airflow limitation; conversely, some patients develop significant airflow limitation without chronic cough and sputum production.[1,8]

NATURAL HISTORY

COPD has a variable natural history and not all individuals follow the same course. However, COPD is generally a progressive disease, especially if a patient's exposure to noxious agents continues. Stopping exposure to these agents, even when significant airflow limitation is present, may result in some improvement in lung function and slow or even halt the progression of the disease. However, once developed, COPD and its comorbidities cannot be cured and thus must be treated continuously. COPD treatment can reduce symptoms, improve quality of life, reduce exacerbations, and possibly reduce mortality[1,8,12]

Spirometric Classification of Severity[1,7,10]

For educational reasons, a simple spirometric classification of disease severity into four stages is recommended. Spirometry is essential for diagnosis and provides a useful description of the severity of pathological changes in COPD. Specific spirometric cutpoints (e.g., post bronchodilator FEV1/FVC ratio < 0.70 or FEV1 < 80, 50, or 30% predicted) are used for purposes of simplicity

Table. 1 GOLD CRITERIA OF COPD SEVERITY[1,3,7,8]

GOLD Stage	Severity	Symptoms	Spirometry
0	At Risk	Chronic cough, sputum production	Normal
I	Mild	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and FEV_1 >80% predicted
IIA	Moderate	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and 50% FEV_1 <80% predicted
III	Severe	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and 30% FEV_1 <50% predicted
IV	Very Severe	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and FEV_1 <30%(**OR**) FEV_1 <50% predicted with respiratory failure or signs of right heart failure

RISK FACTORS

For COPD has grown, so has the recognition that essentially all risk for COPD results from a gene environment interaction. Thus, of two people with the same smoking history, only one may develop COPD due to differences in genetic predisposition to the disease, or in how long they live. Risk factors for COPD may also be related in more complex ways. For example, gender may influence whether a person takes up smoking or experiences certain occupational or environmental exposures; socioeconomic status may be linked to a child's birth weight (as it impacts on lung growth and development); and longer life expectancy will allow greater lifetime exposure to risk factors. Understanding the relationships and interactions among risk factors requires further investigation[1,7,8].

Figure. 2

Risk Factors for COPD

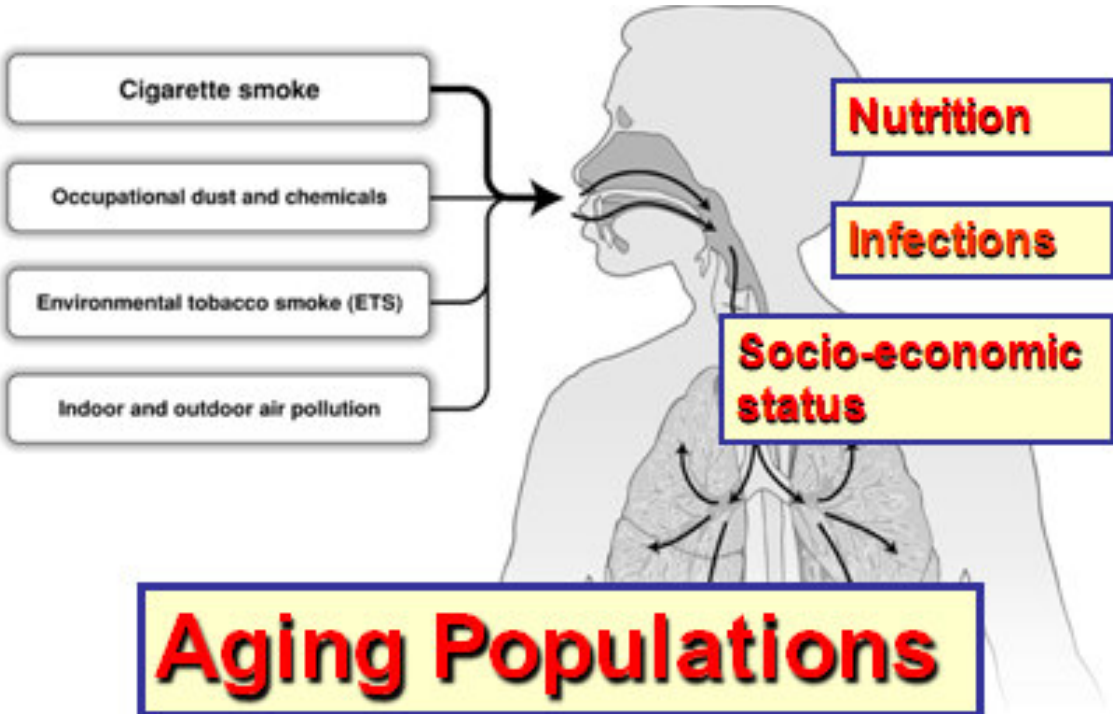


Table.2 RISK FACTORS FOR COPD.[8]

Genes Exposure to particles
Tobacco smoke
Occupational dusts, organic and inorganic
Indoor air pollution from heating and cooking with biomass in poorly vented dwellings
Outdoor air pollution
Lung Growth and Development
Oxidative stress Gender
Age
Respiratory infections
Previous tuberculosis
Socioeconomic status
Nutrition
Comorbidities

Tobacco smoke

Tobacco smoke, which is a mixture of over 4000 chemical constituents, is the most important cause. Amongst males, tobacco smoking is responsible for more than 80% of patients. Both cigarette and 'bidi' smoking are equally responsible Pipe and 'hookah' smoking are also important in causing COPD. There is no reliable information on smoking associated COPD in women in whom the overall prevalence of smoking is very low. Besides active tobacco smoking, exposure to smoking from others i.e. passive

smoking, better termed as Environmental Tobacco Smoke (ETS) exposure, may also play a contributory role especially in nonsmoker individuals including women[1,2,7,9]

Solid fuel combustion

The smoke from combustion of solid fuels such as dried dung, wood and crop residue used for cooking and heating, especially in villages, semi urban and slum areas, is an important cause of pollution of the indoor air. It is responsible for a large number of COPD in the rural inhabitants in general and women in particularly[2,9]

Air pollution

Exhausts from vehicles and industrial units; dusts, fumes and smoke from burning of crop residues in the field constitute important sources of air pollution. Chronic exposure to polluted air is an important cause of chronic respiratory diseases such as the COPD[1,3,8]

Gender

The role of gender in determining COPD risk remains unclear. In the past, most studies showed that COPD prevalence and mortality were greater among men than women. Studies from developed countries show that the prevalence of the disease is now almost equal in men and women, which probably reflects changing patterns of tobacco smoking.[7,13]

Respiratory Infections

Respiratory infections (viral and bacterial) may contribute to the pathogenesis and progression of COPD, and the bacterial colonization associated with airway inflammation, and may also play a significant role in exacerbations. A history of severe childhood respiratory infection has been associated with reduced lung function and increased respiratory symptoms in adulthood. . HIV infection has been shown to accelerate the onset of smoking-related emphysema; HIV induced pulmonary inflammation may play a role in this process. A history of tuberculosis has been found to be associated with airflow obstruction in adults older than 40 years.[1,8]

Asthma

Asthma may be risk factor for the development of COPD, although the evidence is not conclusive. In a report from a longitudinal cohort of the Tucson Epidemiological Study of Airway Obstructive Disease adults with asthma were found to have a twelve-fold higher risk of acquiring COPD over time than those without asthma, after adjusting for smoking[1,13]

PATHOGENESIS

Pathological changes characteristic of COPD are found in the proximal airways, peripheral airways, lung parenchyma, and pulmonary vasculature. These changes

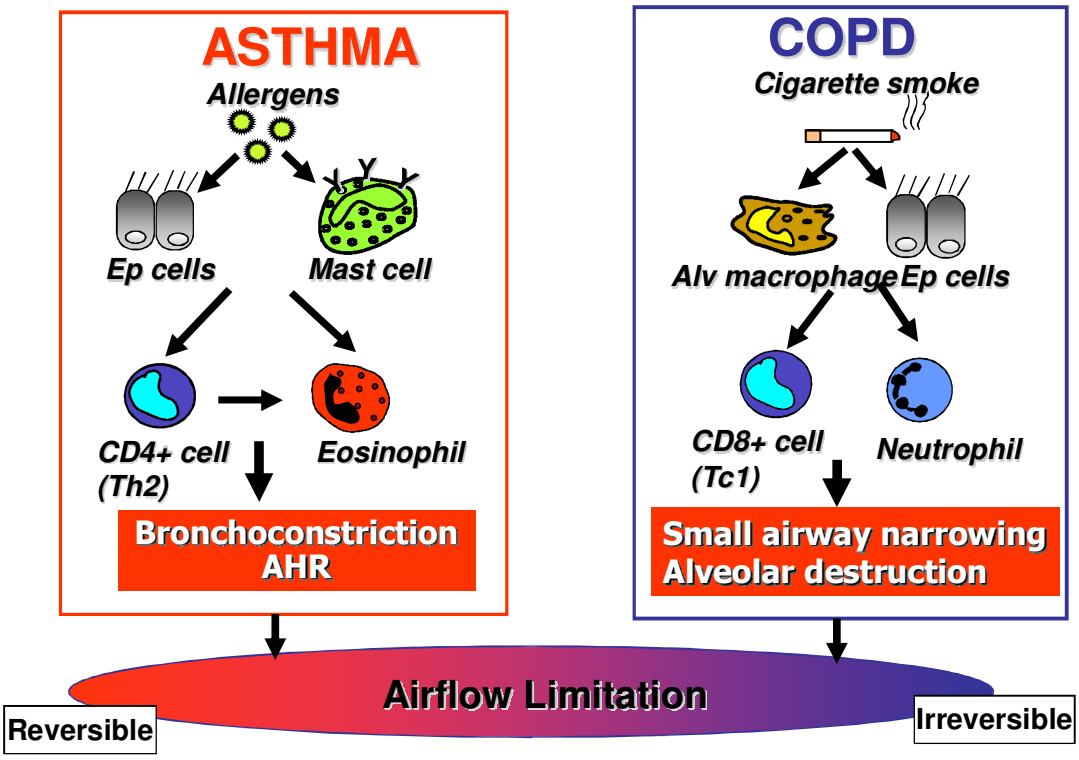


Figure.3 ASTHMA AND COPD[8]

include chronic inflammation, and structural changes resulting from repeated injury and repair.[61]

• Inhaled cigarette smoke and other noxious particles cause lung inflammation, a normal response which appears to be amplified in patients who develop COPD

.• There is a characteristic pattern of inflammation in the lungs of COPD patients, with increased numbers of neutrophils (in the airway lumen), macrophages

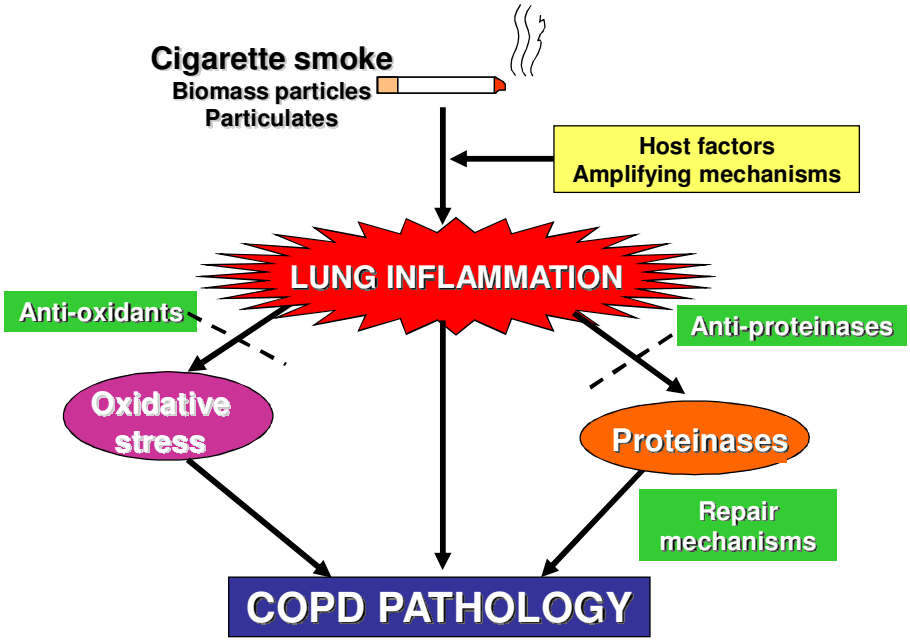


Figure. 4 PATHOLOGY OF COPD

(Airway lumen, airway wall, and parenchyma), and CD8+ lymphocytes (airway wall and parenchyma). The pattern is different from that seen in asthma.[1]

• Lung inflammation is further amplified by oxidative stress and an excess of proteases in the lung Physiological changes characteristic of the disease include mucus hypersecretion, airflow limitation and air trapping (leading to hyperinflation), gas exchange abnormalities, and cor pulmonale.

Exacerbations represent a further amplification of the inflammatory response in the airways of patients with COPD, and may be triggered by infection with bacteria or viruses or by environmental pollutants.[6]

Table 3 PATHOLOGICAL CHANGES COPD[8,61]

Proximal airways (trachea, bronchi > 2 mm internal diameter)

Goblet cells hyperplasia, enlarged submucosal glands (both

leading to mucus hypersecretion), squamous metaplasia of epithelium

Peripheral airways (bronchioles < 2mm i.d.)

Airway wall thickening, peribronchial fibrosis, luminal inflammatory exudate, airway narrowing (obstructive bronchiolitis) Increased inflammatory response and exudate correlated with disease severity

Lung parenchyma (respiratory bronchioles and alveoli)

Destruction of gas exchange air spaces- the respiratory bronchioles, alveolar ducts and alveoli.

*Centrilobular emphysema: dilatation and destruction of respiratory bronchioles; most commonly seen in smokers

*Panacinar emphysema: destruction of alveolar sacs as well as respiratory bronchioles; most commonly seen in alpha-1 antitrypsin deficiency

Pulmonary vasculature

Thickening of intima, endothelial cell dysfunction, smooth muscle pulmonary hypertension

Figure. 5 PATHOGENESIS OF COPD[8]

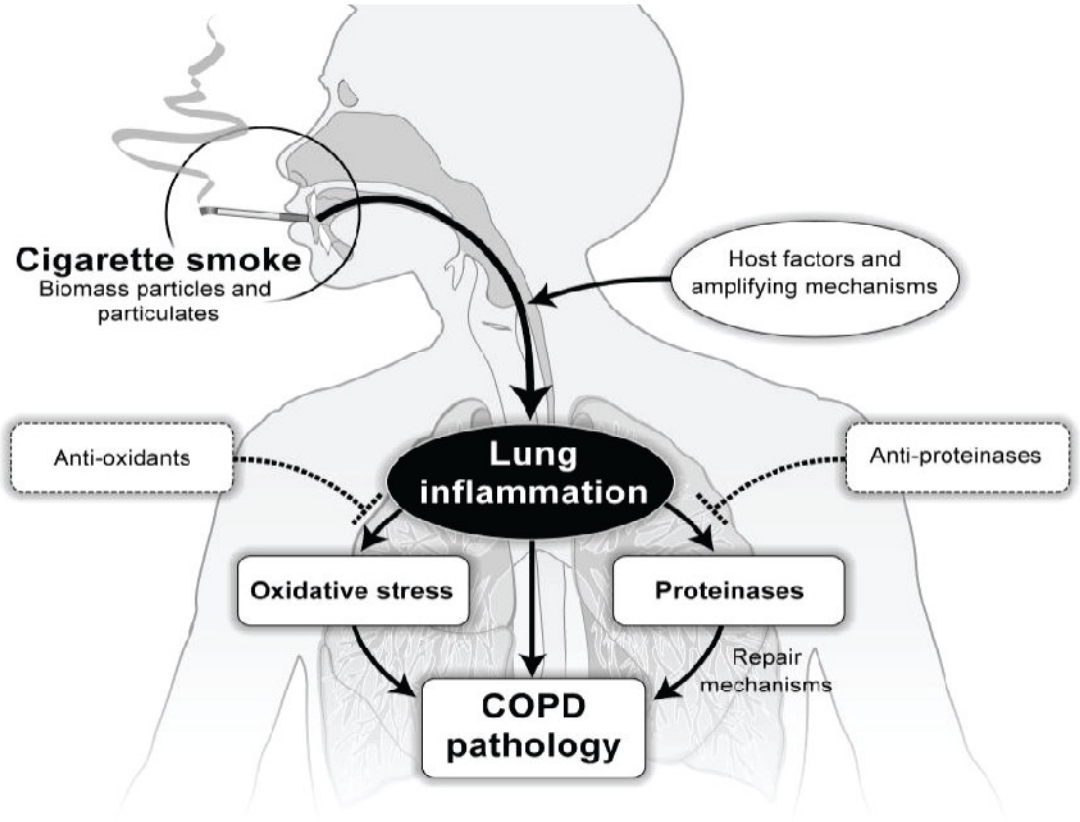


Table 4[6]

Differences in Pulmonary Inflammation Between Asthma and COPD			
	COPD	**Asthma**	Severe Asthma
Cells	Neutrophils ++ Macrophages +++ CD8+ T cells (Tc1)	Eosinophils ++ Macrophages + CD4+ T cells (Th2)	Neutrophils + Macrophages CD4+ T cells (Th2), CD8+ T cells (Tc1)
Key mediators	IL-8 TNF-_, IL-1_, IL-6 No+	Eotaxin IL-4, IL-5, IL-13 NO +++	IL-8 IL-5, IL-13 NO ++
Oxidative stress	+++	+	+++
Site of disease	Peripheral airways Lung parenchyma Pulmonary vessels	Proximal airways	Proximal airways Peripheral airways
Consequences	Squamous metaplasia Mucous metaplasia Small airway fibrosis Parenchymal destruction Pulmonary vascular remodeling	Fragile epithelium Mucous metaplasia Basement membrane Bronchoconstriction	
Response to therapy	Small broncho dilatation response Poor response to steroids	Large broncho dilatation response Good response to steroids	Smaller broncho dilatation response Reduced response to steroids

Pulmonary Hypertension

Mild to moderate pulmonary hypertension may develop late in the course of COPD and is due to hypoxic vasoconstriction of small pulmonary arteries, eventually resulting in structural changes that include intimal hyperplasia and later smooth muscle hypertrophy/ hyperplasia[62]

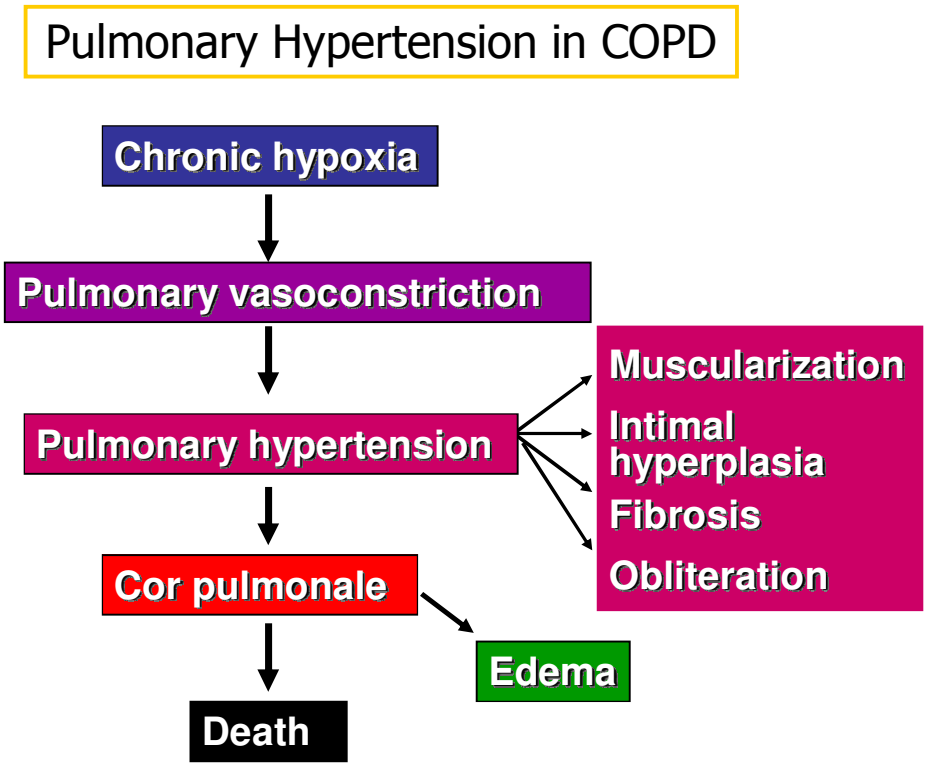


Figure. 6 COPD AND PULMONARY HYPERTENSION

Systemic Features of COPD[6,8]

• Cachexia: loss of fat free mass

• Skeletal muscle wasting: apoptosis, disuse atrophy

• Osteoporosis

• Depression

• Normochromic, normocytic anemia

• Increased risk of cardiovascular disease: associated with CRP

MANAGEMENT

Management of Mild to Moderate COPD (*Stages I and II*) involves the avoidance of risk factors to prevent disease progression and pharmacotherapy as needed to control symptoms. Severe (*Stage III*) and Very Severe (*Stage IV*) COPD often require the integration of several different disciplines, a variety of treatment approaches, and a commitment of the clinician to the continued support of the patient as the illness progresses[1,3,6]. In addition to patient education, health advice, and pharmacotherapy, COPD patients may require specific counseling about smoking cessation, instruction in physical exercise, nutritional advice, and continued nursing support. Not all approaches are needed for every patient, and assessing the potential benefit of each approach at each stage of the illness is a crucial aspect of effective disease management. While disease prevention is the ultimate goal, once COPD has been diagnosed, effective management should be aimed at the following goals[8]:

• Relieve symptoms

• Prevent disease progression

• Improve exercise tolerance

• Improve health status

• Prevent and treat complications

• Prevent and treat exacerbations

• Reduce mortality

A clinical diagnosis of COPD should be considered in any patient who has dyspnoea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease. The diagnosis should be confirmed by spirometry. For the diagnosis and assessment of COPD, spirometry is the gold standard as it is the most reproducible, standardized, and objective way of measuring airflow limitation. The presence of a post bronchodilator FEV1/FVC < 0.70 confirms the presence of airflow limitation that is not fully reversible[1.8]. Measurement of arterial blood gas tensions should be considered in all patients with FEV1 < 50% predicted or clinical signs suggestive of respiratory failure or right heart failure[4].

(1) Assess and Monitor Disease;

(2) Reduce Risk Factors;

(3) Manage Stable COPD; and

(4) Manage Exacerbations.

Dyspnoea. Dyspnoea, the hallmark symptom of COPD, is the reason most patients seek medical attention and is a major cause of disability and anxiety associated with the disease. Typical COPD patients describe their dyspnoea as a sense of increased effort to breathe, heaviness, air hunger, or gasping[5,6]. It is often possible to distinguish the breathlessness of COPD from that due to other causes by analysis of the terms used, although there is considerable overlap with descriptors of bronchial asthma. Initially,

breathlessness is only noted on unusual effort (e.g., walking or running up a flight of stairs) and may be avoided entirely by appropriate behavioral change. As lung function deteriorates, breathlessness becomes more intrusive, and patients may notice that they are unable to walk at the same speed as other people of the same age or carry out activities. Eventually, breathlessness is present during everyday activities (e.g., dressing, washing) or at rest[2]

Cough. Chronic cough, often the first symptom of COPD to develop, is often discounted by the patient as an expected consequence of smoking and/or environmental exposures. Initially, the cough may be intermittent, but later is present every day, often throughout the day. The chronic cough in COPD may be unproductive. In some cases, significant airflow limitation may develop without the presence of a cough[1,4,6].

Sputum production. COPD patients commonly raise small quantities of tenacious sputum after coughing bouts. Regular production of sputum for 3 or more months in 2 consecutive years (in the absence of any other conditions that may explain it) is the epidemiological definition of chronic bronchitis, but this is a somewhat arbitrary definition that does not reflect the range of sputum production in COPD patients. Sputum production is often difficult to evaluate because patients may swallow sputum rather than expectorate it, a habit subject to significant cultural and gender variation. Patients producing large volumes of sputum may have the underlying bronchiectasis. The presence of purulent sputum reflects an increase in inflammatory mediators, and its development may identify the onset of an exacerbation[5,61]

Wheezing and chest tightness. Wheezing and chest tightness are nonspecific symptoms that may vary between days, and over the course of a single day. These symptoms may be present in *Stage I: Mild COPD*, but are more characteristic of asthma or *Stage III: Severe COPD* and *Stage IV: Very Severe COPD*. Audible wheeze may arise at a laryngeal level and need not be accompanied by auscultatory abnormalities. Alternatively, widespread inspiratory or expiratory wheezes can be present on listening to the chest. Chest tightness often follows exertion, is poorly localized, is muscular in character, and may arise from isometric contraction of the intercostals muscles. An absence of wheezing or chest tightness does not exclude a diagnosis of COPD, nor does their presence confirm a diagnosis of asthma.[8]

Medical History

A detailed medical history of a new patient known or thought to have COPD should assess the patient's exposure to risk factors, such as smoking and occupational or environmental exposures Past medical history, including asthma, allergy, sinusitis, or nasal polyps, respiratory infections in childhood; other respiratory diseases. Family history of COPD or other chronic respiratory disease. Pattern of symptom development: COPD typically develops in adult life and most patients are conscious of increased breathlessness, more frequent winter colds, and some social restriction for a number of years before seeking medical help.

• History of exacerbations or previous hospitalizations for respiratory disorder: Patients may be aware of periodic worsening of symptoms even if these episodes have not been identified as exacerbations of COPD.

• Presence of comorbidities, such as heart disease, malignancies, osteoporosis, and musculoskeletal disorders, which may also contribute to restriction of activity.

•Appropriateness of current medical treatments: For example, betablockers commonly prescribed for heart disease are usually contraindicated in COPD.

• Possibilities for reducing risk factors, especially smoking cessation

Physical Examination

Though an important part of patient care, physical examination is rarely diagnostic in COPD. Detection has a relatively low sensitivity and specificity. A number of physical signs may be present in COPD, but their absence does not exclude the diagnosis.

• Central cyanosis, or bluish discoloration of the mucosal membranes,

• Common chest wall abnormalities, which reflect the pulmonary hyperinflation seen in COPD, include relatively horizontal ribs, “barrel-shaped” chest, and protruding abdomen.

• Flattening of the hemidiaphragms may be associated with paradoxical indrawing of the lower rib cage on inspiration, and widening of the xiphisternal angle.

• Resting respiratory rate is often increased COPD patients often have resting muscle activation while lying supine. Use of the scalene and sternocleidomastoid muscles is a further indicator of respiratory distress.

• Ankle or lower leg edema can be a sign of right heart failure. . Patients with COPD often have reduced breath sounds, but this finding is not sufficiently characteristic to make the diagnosis[5].

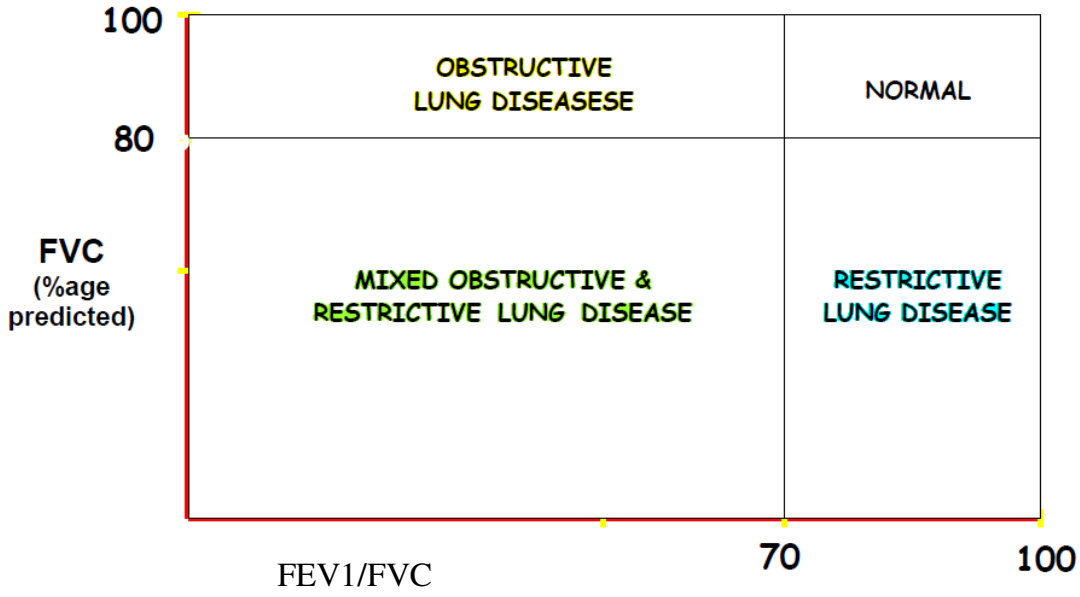
• The presence of wheezing during quiet breathing is a useful pointer to airflow limitation. However, wheezing heard only after forced expiration has not been validated as a diagnostic test for COPD.[1]

• Inspiratory crackles occur in some COPD patients but are of little help diagnostically.[6]

Measurement of Airflow Limitation (Spirometry)[2,8]

Spirometry should be undertaken in all patients who may have COPD. It is needed to make a confident diagnosis of COPD and to exclude other diagnoses that may present with similar symptoms. It is the best standardized, most reproducible, and most objective measurement of airflow limitation available. Good quality spirometric measurement is possible and all health care workers who care for COPD patients should have access to spirometry.[8,10]

CLASSIFICATION OF OBSTRUCTIVE OR RESTRICTIVE OR MIXED AIRWAY DISEASE BASED ON FVC AND FEV1 VALUES[10]



Spirometry should measure the volume of air forcibly exhaled from the point of maximal inspiration (forced vital capacity, FVC) and the volume of air exhaled during the first second of this maneuver (forced expiratory volume in one second, FEV1)[10], and the ratio of these two measurements (FEV1/FVC) should be calculated. Spirometry measurements are evaluated by comparison with reference values based on age, height, sex, and race[8,10]

Performance

• Spirometry should be performed using techniques that meet published standards.

• The expiratory volume/time traces should be smooth and free from irregularities. • The recording should go on long enough for a volume plateau to be reached, which may take more than 15 seconds in severe disease[8,10,18].

• Both FVC and FEV1 should be the largest value obtained from any of 3 technically satisfactory curves and the FVC and FEV1 values in these three curves should vary by no more than 5% or 100 ml, whichever is greater[23].

• The FEV1/FVC ratio should be taken from the technically acceptable curve with the largest sum of FVC and FEV1.

Evaluation

• Spirometry measurements are evaluated by comparison of the results with appropriate reference values based on age, height, sex, and race [10]

• The presence of a post bronchodilator FEV1/FVC<0.70 confirms the presence of airflow limitation that is not fully reversible[18,23]

Chest X-ray. An abnormal chest X-ray is seldom diagnostic in COPD unless obvious bullous disease is present, but it is valuable in excluding alternative diagnoses and establishing the presence of significant comorbidities such as cardiac failure. Radiological changes associated with COPD include signs of hyperinflation (flattened

diaphragm on the lateral chest film, and an increase in the volume of the retrosternal air space), hyperlucency of the lungs, and rapid tapering of the vascular markings. Computed tomography (CT) of the chest is not routinely recommended. However, when there is doubt about the diagnosis of COPD, high resolution CT (HRCT) scanning might help in the differential diagnosis.[4,6]

Arterial blood gas measurement. In advanced COPD, measurement of arterial blood gases is important. This test should be performed in the stable patients with FEV1 < 50% predicted or with clinical signs suggestive of respiratory failure or right heart failure. Several considerations are important to ensure accurate test results. [66]

Table 5. EMPHYSEMA VS CHRONIC BRONCHITIS[4]

	Type A: Pink Puffer (Emphysema Predominant)	**Type B: Blue Bloater (Bronchitis Predominant)**
History and physical examination	Major complaint is dyspnoea, often severe, usually presenting after age 50. Cough is rare, with scant clear, mucoid sputum. Patients are thin, with recent weight loss common. They appear uncomfortable, with evident use of accessory muscles of respiration. Chest is very quiet without adventitious sounds. No peripheral edema.	Major complaint is chronic cough, productive of mucopurulent sputum, with frequent exacerbations due to chest infections. Often presents in late 30s and 40s. Dyspnoea usually mild, but may note limitations to exercise. Chest is noisy, with rhonchi invariably present; wheezes are common.
Laboratory studies	Hemoglobin usually normal. PaO_2 normal to slightly reduced but SaO_2 normal at rest. $PaCO_2$ normal to slightly reduced. Chest radiograph shows hyperinflation with flattened diaphragms. Vascular markings are diminished, particularly at the apices.	Hemoglobin usually elevated. PaO_2 reduced and $PaCO_2$ slightly to markedly elevated. Chest radiograph shows increased interstitial markings ("dirty lungs"), especially at bases. Diaphragms are not flattened.
Pulmonary function tests	Airflow obstruction ubiquitous. Total lung capacity increased, sometimes markedly so. DL_{CO} reduced. Static lung compliance increased.	Airflow obstruction ubiquitous. Total lung capacity generally normal but may be slightly increased. DL_{CO} normal. Static lung compliance normal.
Special evaluations		
V/Q matching	Increased ventilation to high areas, i.e., high dead space ventilation.	Increased perfusion to low areas.
Hemodynamics	Cardiac output normal to slightly low. Pulmonary artery pressures mildly elevated and increase with exercise.	Cardiac output normal. Pulmonary artery pressures elevated, sometimes markedly so, and worsen with exercise.
Nocturnal ventilation	Mild to moderate degree of oxygen desaturation not usually associated with obstructive sleep apnea.	Severe oxygen desaturation, frequently associated with obstructive sleep apnea.
Exercise ventilation	Increased minute ventilation for level of oxygen consumption. PaO_2 tends to fall, $PaCO_2$ rises slightly	Decreased minute ventilation for level of oxygen consumption. PaO_2 may rise; $PaCO_2$ may rise

Monitor Disease Progression and Development of

Complications

COPD is usually a progressive disease. Lung function can be expected to worsen over time. As at the initial assessment, follow-up visits should include a physical examination and discussion of symptoms, particularly any new or worsening symptoms[8].

Pulmonary function. A patient's decline in lung function is best tracked by periodic spirometry measurements although useful information about lung function decline is unlikely from spirometry measurements performed more than once a year[10]

Electrocardiography findings

ECG may show sinus tachycardia. In advanced disease, chronic pulmonary hypertension may produce electrocardiographic abnormalities typical of cor pulmonale .Supraventricular arrhythmias (multifocal atrial tachycardia, atrial flutter, and atrial fibrillation) and ventricular irritability also occur[69]

Diagnosis of right heart failure or cor pulmonale.

Elevation of the jugular venous pressure and the presence of pitting ankle edema are often the most useful findings suggestive of cor pulmonale in clinical practice. Firm diagnosis of cor pulmonale can be made through a number of investigations, including radiography, electrocardiography, echocardiography[67,69]

Hematocrit. Polycythemia can develop in the presence of arterial hypoxemia, especially in continuing smokers43, and can be identified by Hematocrit > 55%. Anemia is more prevalent than previously thought, affecting almost a quarter of COPD patients in one hospital series. A low Hematocrit indicates a poor prognosis in COPD patients receiving long-term oxygen treatment[1,7].

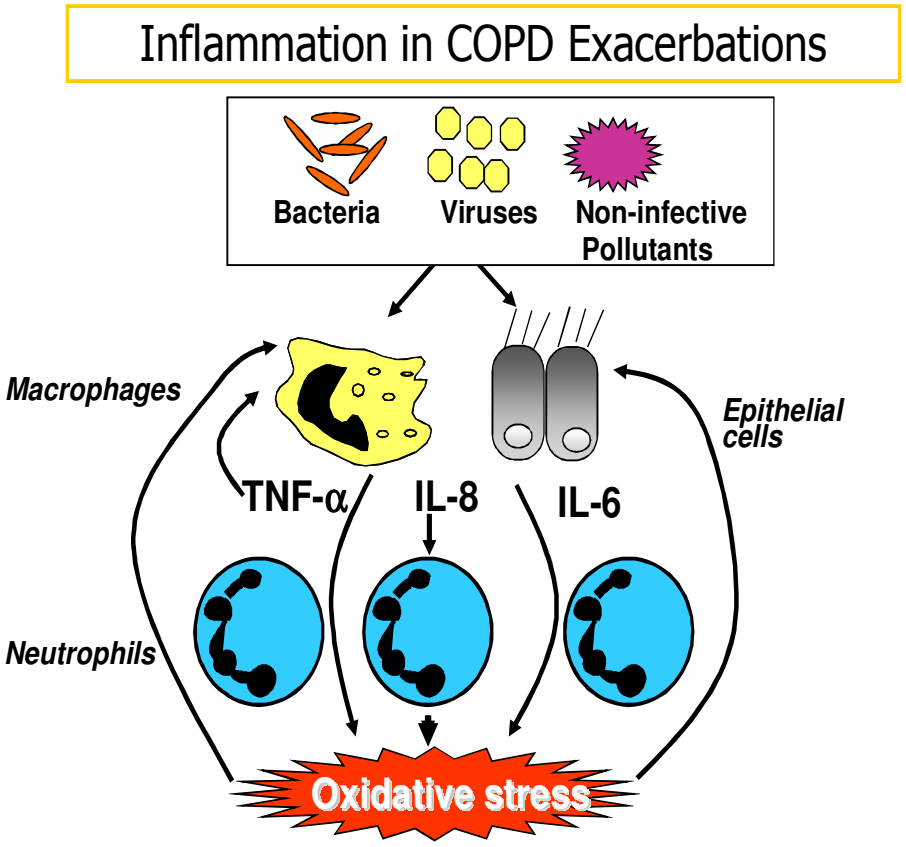
Monitor Pharmacotherapy and Other Medical Treatment

In order to adjust therapy appropriately as the disease progresses, each follow-up visit should include a discussion of the current therapeutic regimen. Dosages of various medications, adherence to the regimen, inhaler technique, effectiveness of the current regime at controlling symptoms, and side effects of treatment should be monitored[8,10].

Monitor Exacerbation History

During periodic assessments, should question the patient and evaluate any records of exacerbations, both self treated and those treated by other health care providers. Frequency, severity, and likely causes of exacerbations should be evaluated. Frequency, severity, likely causes of exacerbations. Increased sputum volume, acutely worsening dysponea, and the presence of purulent sputum should be noted. Severity can be estimated by the increased need for bronchodilator medication or glucocorticosteroids and by the need for antibiotic treatment[1,4,8]

Figure.7



Monitor Comorbidities

Comorbidities are common in COPD. Some may be an indirect result of COPD, arising independently but more likely to occur when COPD is present, e.g., ischemic heart disease, bronchial carcinoma, osteoporosis, and depression.. Other comorbid conditions may coexist with COPD because they become prevalent as part of the aging process, e.g., arthritis, diabetes, reflux esophagitis and depression Reduction of total personal exposure to tobacco smoke, occupational dusts and chemicals, and indoor and outdoor air pollutants are important goals to prevent the onset and progression of COPD[8].

• Smoking cessation is the single most effective and cost effective intervention in most people to reduce the risk of developing COPD and stop its progression strategies aimed at reducing the burden of inhaled particles and gases[40].

• Reducing the risk from indoor and outdoor air pollution is feasible and requires a combination of public policy and protective steps taken by individual patients[1,8].

Smoking Prevention

Comprehensive tobacco control policies and programs with clear, consistent, and repeated nonsmoking messages should be delivered through every feasible channel, including health care providers, community activities, schools, and radio, television, and print media. National and local campaigns should be undertaken to reduce exposure to tobacco smoke in public forums. Such bans are proving to be workable and to result in measurable gains in respiratory health. Legislation to

establish smoke free schools, public facilities, and work environments should be developed and implemented by government officials and public health workers, and encouraged by the public. Smoking prevention programs should target all ages, including young children, adolescents, young adults, and pregnant women. Interventions to prevent smoking uptake and maximize cessation should be implemented at every level of the health care system. Physicians and public health officials should encourage smoke free homes. The first exposure to cigarette smoke may begin in utero when the fetus is exposed to bloodborne metabolites from the mother. Education to reduce in utero risks for unborn children is also of great importance to prevent the effects of maternal smoking in reducing lung growth and causing airways disease in early and later life[8].

BRIEF STRATEGIES TO HELP THE PATIENT WILLING TO QUIT SMOKING[8]

- ASK Systematically identify all tobacco users at every visit.
- ADVISE Strongly urge all tobacco users to quit.
- ASSESS Determine willingness to make a quit attempt
- ASSIST Aid the patient in quitting
- ARRANGE Schedule follow-up contact

PHARMACOLOGICAL TREATMENT

Bronchodilators

Medications that increase the FEV1 or change other spirometric variables, usually by altering airway smooth muscle tone, are termed bronchodilators, since the improvements in expiratory flow reflect widening of the airways rather than changes in lung elastic recoil. Such drugs improve emptying of the lungs, tend to reduce dynamic hyperinflation at rest and during exercise, and improve exercise performance [1,3].

β2-agonists. The principal action of β2-agonists is t. relax airway smooth muscle by stimulating β2-adrenergic receptors, which increases cyclic AMP and produces functional antagonism to bronchoconstriction. Oral therapy is slower in onset and has more side effects than inhaled treatment

Short-acting

Fenoterol, Levalbuterol, Salbutamol, Terbutaline

Long-acting

Formoterol ,Arformoterol, Indacaterol[8] ,Salmeterol

Anticholinergics. The most important effect of anticholinergic medications, such as ipratropium, oxitropium and tiotropium bromide, in COPD patients appears to be blockage of acetylcholine's effect on M3 receptors. Current short-acting drugs also block M2 receptors and modify transmission at the pre-ganglionic junction, although these effects appear less important in COPD. The long-acting anticholinergic tiotropium has a

pharmacokinetic selectivity for the M3 and M1 receptors. The bronchodilating effect of short-acting inhaled anticholinergics lasts longer than that of short-acting β2-agonists [1,8]

Short-acting

Ipratropium bromide ,Oxitropium bromide

Long-acting

Tiotropium

Combination short-acting β2-agonists plus anticholinergic in one inhaler

Fenoterol/Ipratropium

Salbutamol/Ipratropium

Methylxanthines. Controversy remains about the exact effects of xanthine derivatives. They may act as nonselective phosphodiesterase inhibitors, but have also been reported to have a range of no bronchodilator actions, the significance of which is disputed

Aminophylline, Theophylline

Glucocorticosteroids

The effects of oral and inhaled glucocorticosteroids in COPD are much less dramatic than in asthma, and their role in the management of stable COPD is limited to specific indications

Inhaled glucocorticosteroids.

Most studies have shown that regular treatment with inhaled glucocorticosteroids does not modify the long-term decline of FEV1 in patients with COPD

Beclomethasone

Budesonide

Fluticasone propionate

***Combination inhaled glucocorticosteroid/bronchodilator therapy*:**

An inhaled glucocorticosteroid combined with a long-acting β2-agonist is more effective than the individual components in reducing exacerbations and improving lung function and health status

Formoterol/Budesonide

Salmeterol/Fluticasone propionate

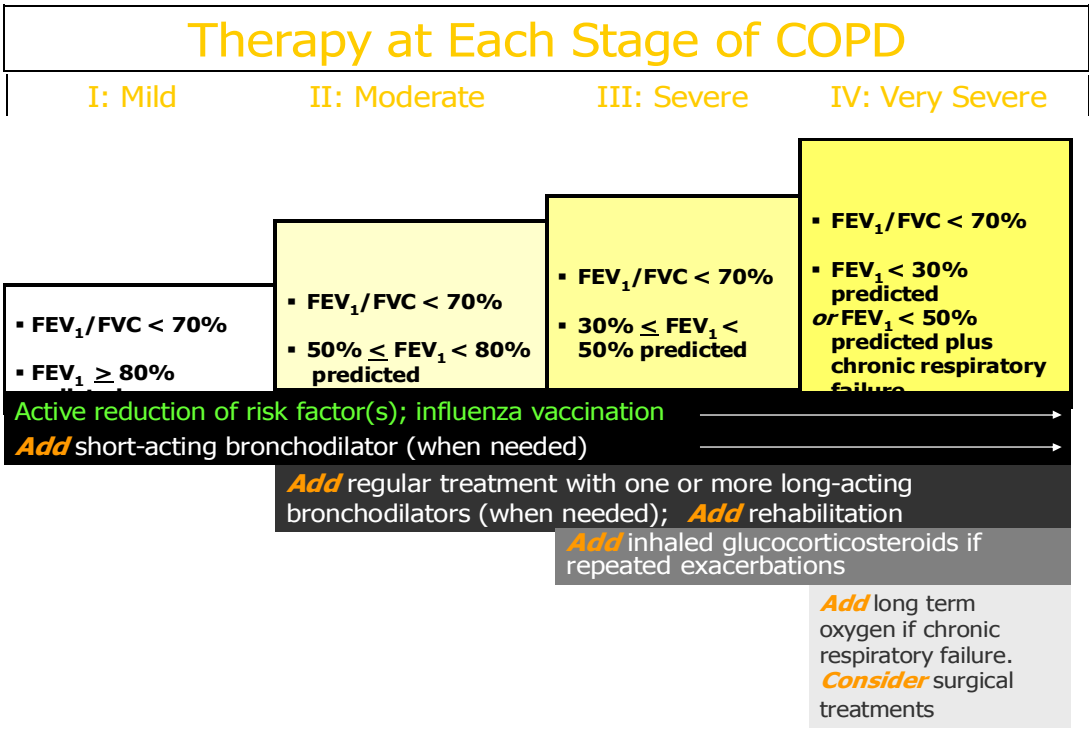
Oral glucocorticosteroids:

short-term. Many existing COPD guidelines recommend the use of a short course (two weeks) of oral glucocorticosteroids to identify COPD patients who might benefit from long-term treatment with oral or inhaled glucocorticosteroids.

Prednisone

Methyl-prednisolone

Table. 6



VACCINES

Influenza vaccines can reduce serious illness and death in COPD patients by about 50%

Rehabilitation:

All COPD patients benefit from exercise training programs, improving with respect to both exercise tolerance and symptoms of dyspnoea and fatigue.

Mucolytic (mucokinetic, mucoregulator) agents

(ambroxol, erdosteine, carbocysteine, iodinated glycerol). The regular use of mucolytics in COPD has been evaluated in a number of long-term studies with controversial results. Although a few patients with viscous sputum may benefit from mucolytics

Oxygen Therapy

Oxygen therapy, one of the principal nonpharmacologic treatments for patients with *Stage IV: Very Severe COPD*, can be administered in three ways: long-term continuous therapy, during exercise, and to relieve acute dyspnoea. The primary goal of oxygen therapy is to increase the baseline PaO2 to at least 8.0 kPa (60 mm Hg) at sea level and rest, and/or produce an SaO2 at least 90%, which will preserve vital organ function by ensuring adequate delivery of oxygen[1,6,8]

Surgical Treatments

Bullectomy. Bullectomy is an older surgical procedure for bullous emphysema. Removal of a large bulla that does not contribute to gas exchange decompresses the adjacent lung parenchyma. Bullectomy can be performed thoracoscopically. In carefully selected patients, this procedure is effective in reducing dyspnoea and improving lung function[3,8]

Lung transplantation. In appropriately selected patients with very advanced COPD, lung transplantation has been shown to improve quality of life and functional capacity. Criteria for referral for lung transplantation include FEV1 < 35% predicted, PaO2 <7.38.0 kPa

(5560 mm Hg), PaCO2 > 6.7 kPa (50 mm Hg), and secondary pulmonary hypertension [1,3,8]

MANAGE EXACERBATIONS

The most common causes of an exacerbation are infection of the tracheobronchial tree and air pollution, but the cause of about one third of severe exacerbations cannot be identified.

• Inhaled bronchodilators (particularly inhaled β2-agonists with or without anticholinergics) and oral glucocorticosteroids are effective treatments for exacerbations of COPD.

• Patients experiencing COPD exacerbations with clinical signs of airway infection (e.g., increased sputum purulence) may benefit from antibiotic treatment.

• Noninvasive mechanical ventilation in exacerbations improves respiratory acidosis, increases pH, decreases the need for endotracheal intubation, and reduces PaCO2, respiratory rate, severity of breathlessness, the length of hospital stay, and mortality

• Medications and education to help prevent future exacerbations should be considered as part of follow-up, as exacerbations affect the quality of life and prognosis of patients with COPD

MATERIALS AND METHODS

MATERIALS AND METHODS

SETTING

The study was conducted in Annal Gandhi Memorial Government Hospital, Trichy which is attached to K. A. P. Viswanatham Government Medical College, Trichy.

DESIGN OF STUDY

It was an observational type of study. Interview technique was used to collect information on a predesigned proforma.

PERIOD OF STUDY

It was conducted in a time period from January 2011 to October 2011.

SAMPLE SIZE

Hundred cases of Chronic Obstructive Pulmonary Disease.

SELECTION OF STUDY SUBJECTS

COPD patients above 40 years of age, admitted in medical wards with the diagnosis of Chronic Obstructive Pulmonary Disease.

INCLUSION CRITERIA

Adult males and females admitted in the medical wards with symptoms suggestive of airway obstruction of more than 2 years duration and in who clinical diagnosis of chronic obstructive pulmonary disease was made were included in the study.

All these patients were subjected to clinical examination, ECG, chest X-ray, pulmonary function testing, pulse oximetry and Hematocrit analysis

On spirometry the presence of COPD was diagnosed by post bronchodilator values of

(I) Forced expiratory volume in first second / Forced vital capacity (FEV1/FVC) less than 70%.

All patients were clinically stable at the time of conducting pulmonary function test.

EXCLUSION CRITERIA

Cases which excluded from the study were patients with primary diagnosis of bronchial asthma, pulmonary tuberculosis, Bronchiectasis, cases of sleep apnoea syndromes and patients with post infarction failure.

OBSERVATION & RESULTS

OBSERVATIONS AND RESULTS

100 cases of COPD were studied; the results are tabulated as follows:

Table.7 AGE WISE DISTRIBUTION

AGE	MALE	%	FEMALE	%	TOTAL	%
41-50	8	8.6	0		8	8
51-60	22	24	4	50	26	26
61-70	46	50	0		46	46
71-80	16	17.4	2	25	18	18
81-90	0	0	2	25	2	2
TOTAL	92	100	8	100	100	100

From the above table it can be observed that the maximum case among males were between 61-70 years of age constituting 50% and the Minimum number cases were in the age group of 81-90 years being 0%. Among females, number of cases in 51-60 years was 4 constituting 50% and in the age group of 71-80 and 81-90 were 2 cases each constituting of 25% each respectively. Both sexes put together the maximum cases were in age group of 61-70 years constitutes 46 % and minimum in the age group of 81-90 years constituting only 2%.

CHART SHOWING AGE DISTRIBUTION IN THE STUDIED POPULATION

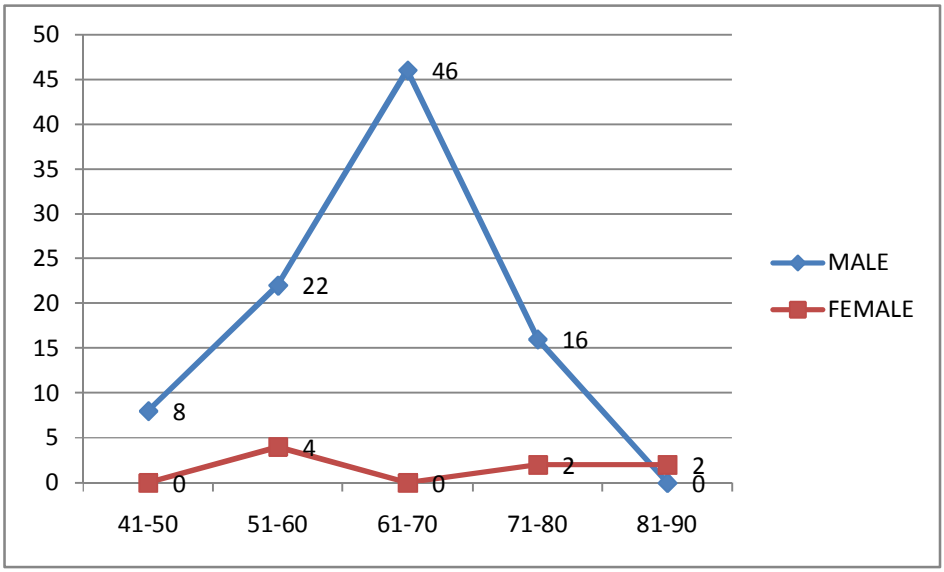


Table 8. SEX DISTRIBUTION IN STUDIED POPULATION

SEX	NO OF CASES	PERCENTAGE
MALE	92	92
FEMALE	8	8
TOTAL	100	100

PIE CHART SHOWING SEX DISTRIBUTION

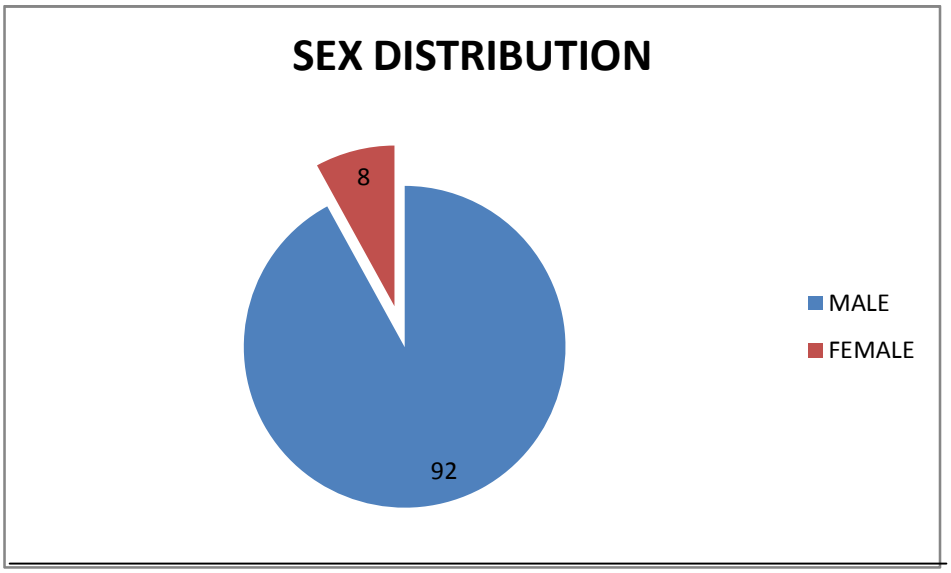
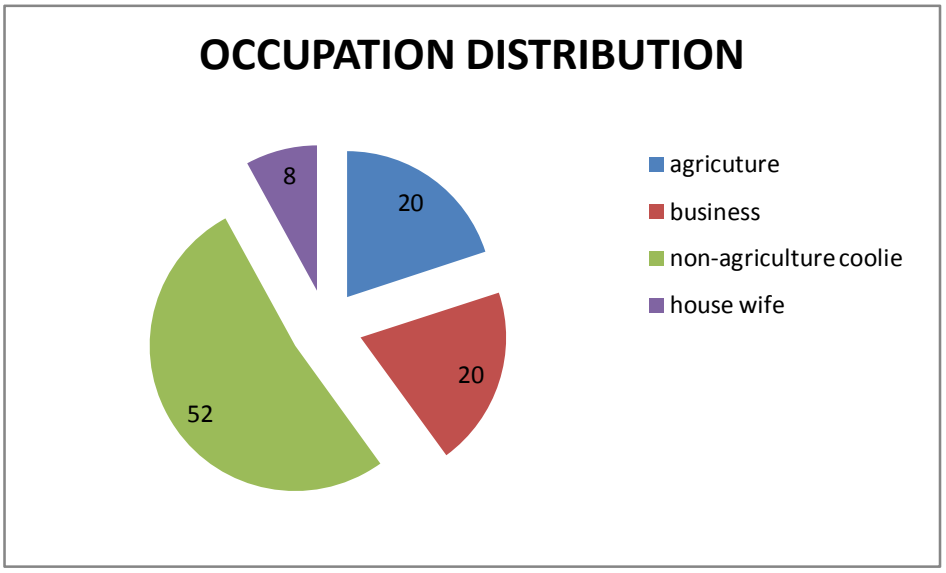


Table 9. OCCUPATION OF THE PATIENTS

Occupation	No of cases	percentage
Agriculture	20	20
Business	20	20
Non-agricultural coolie	52	52
House wife	8	8



In the present study group majority of the patients were non- agricultural coolies accounting for 54%. The remaining were businessmen and agriculture 20% each. House wives were accounting for 8%.

Table 10. RURAL- URBAN DISTRIBUTION

	Cases	Percentage
RURAL	38	38
URBAN	62	62
TOTAL	100	100

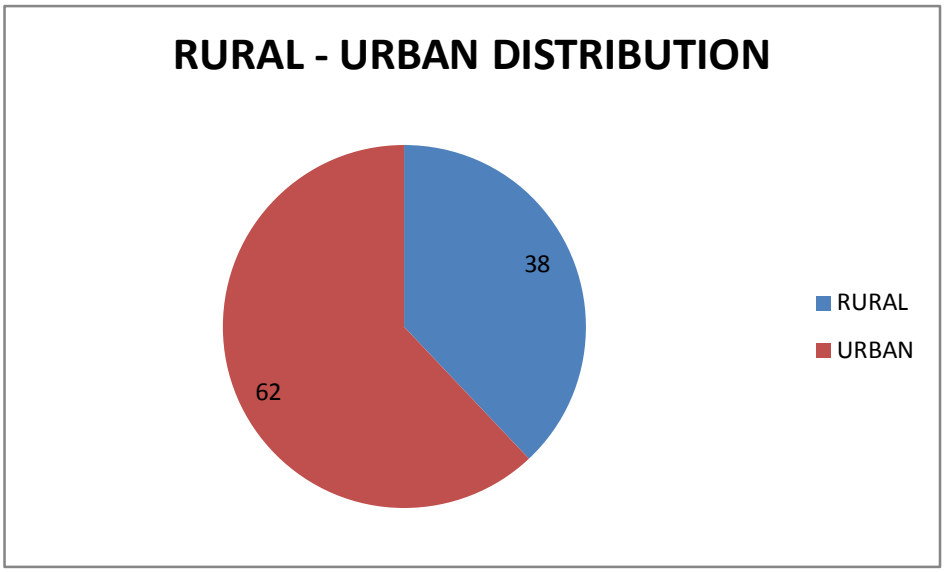


Table.11. DURATION OF ILLNESS

DURATION IN YRS	MALE	FEMALE	TOTAL	PERCENT
2-5	44	0	44	44
6-10	34	2	36	36
11-15	12	6	18	18
16-20	2	0	4	4

Majority of people in the present study group belonged to more than 2-5 years duration of illness.

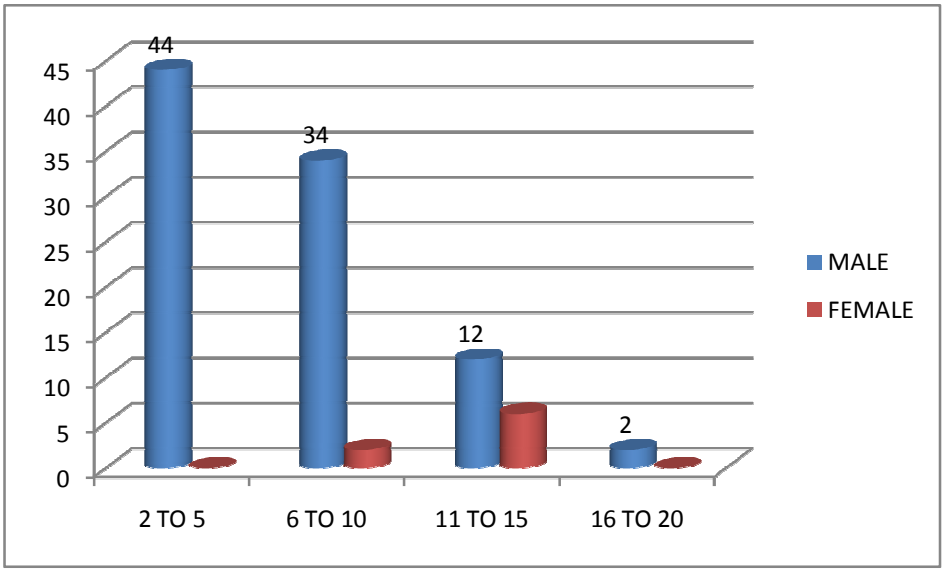
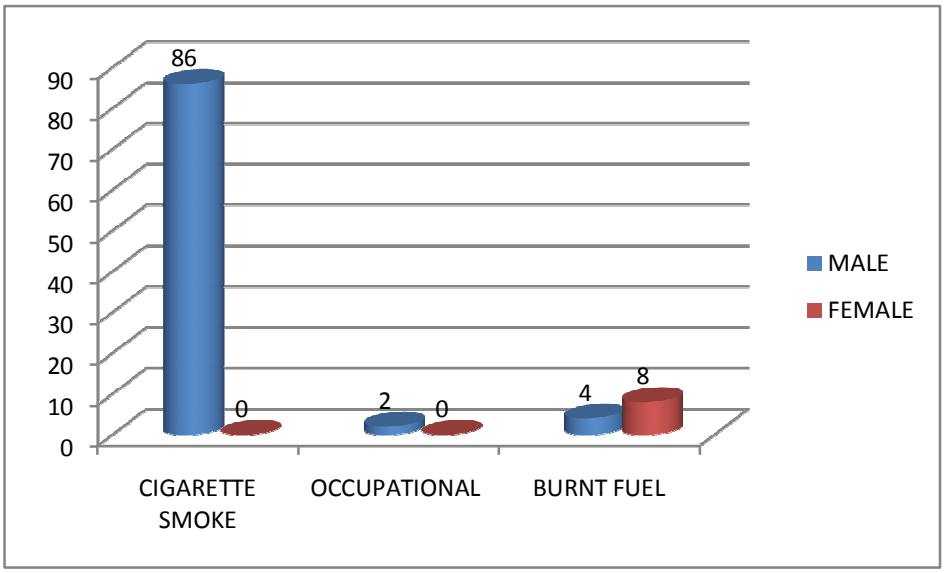


Table 12. RISK FACTOR EXPOSURE

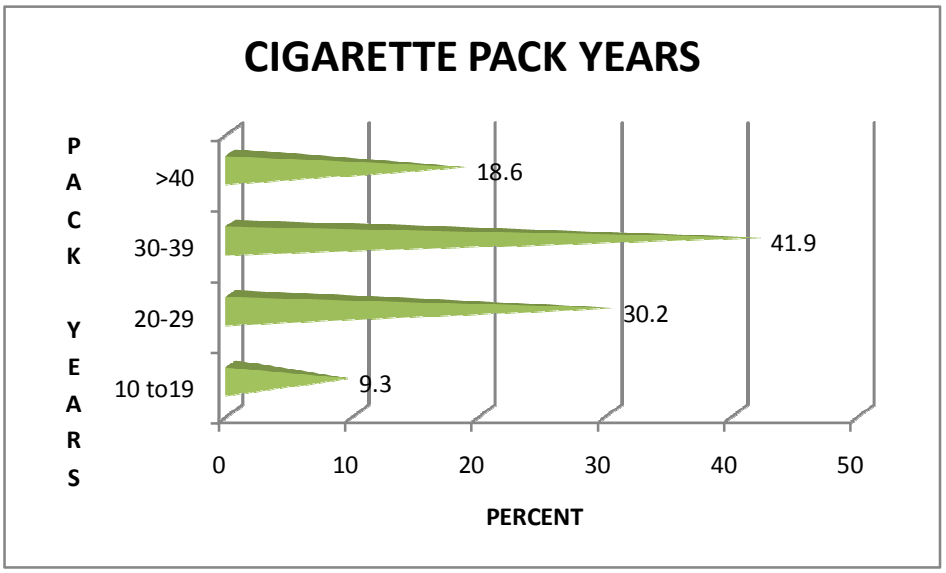
RISK FACTOR	MALE	FEMALE	TOTAL	PERCENTAGE
CIGARETTE SMOKE	86	0	86	86
OCCUPATIONAL	2	0	2	2
BURNT FUEL	4	8	12	12



All the male patients were smokers; in female H/o exposure to smoke of burnt fuels was present in all case.

Table 13. DURATION OF SMOKING HABITS

PACK YEARS	NO OF PATIENTS	PERCENTAGE
10-19	8	9.3
20-29	26	30.2
30-39	36	41.9
>40	16	18.6
TOTAL	86	100

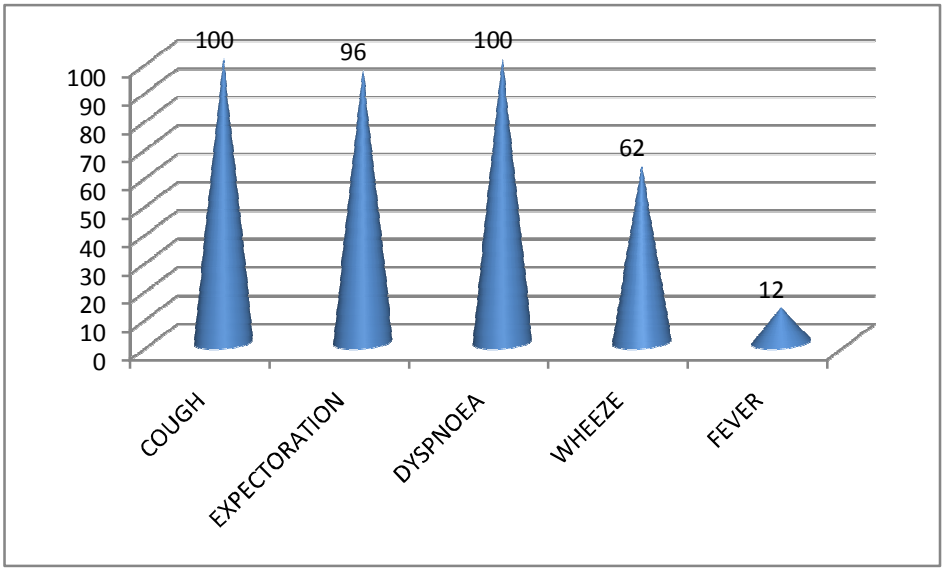


Most of the patients had more than 20 years and majority were in

30-50 pack year exposure duration.

Table-14: PRESENTING SYMPTOMS

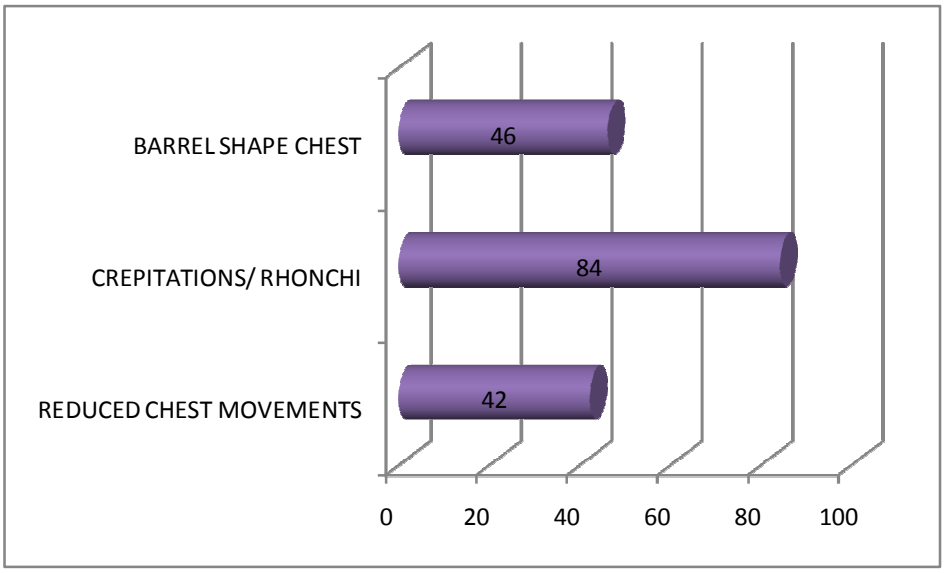
SYMPTOM	NO OF PATIENTS	PERCENTAGE
COUGH	100	100
EXPECTORATION	96	96
DYSPNOEA	100	100
WHEEZE	62	62
FEVER	12	12



All the patients presented with cough and dyspnoea. Expectoration and wheezing was present in majority of the patients and fever was present among a small percentage (12%) of patients. Majority of patients were in the stable state.

Table-15: RESPIRATORY SIGNS

SIGN	NO OF PATIENTS	PERCENTAGE
REDUCED CHEST MOVEMENTS	42	42
CREPITATIONS/ RHONCHI	84	84
BARREL SHAPE CHEST	46	46



Most of patients presented with obesity, short neck and Nicotine stain in lips.

Table-16: CHEST X RAY FINDINGS

X-RAY FINDINGS	NO OF CASES	PERCENTAGE
CHRONIC BRONCHITIS	32	32
EMPHYSEMA	28	28
CHRONIC BRONCHITIS+EMPHYSEMA	18	18
NORMAL	22	22

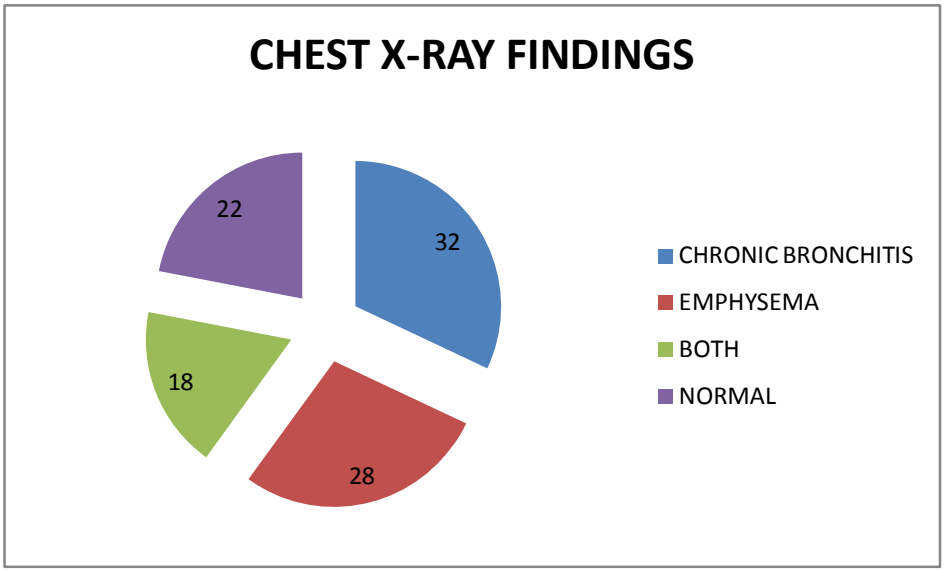


Table-17: ECG FINDINGS

FINDINGS IN ECG	No of cases	%
LOW VOLTAGE COMPLEXES	24	24
SINUS TACHYCARDIA	30	30
L 1 SIGN	4	4
RIGHT ATRIAL ENLARGEMENT	8	8
RIGHT VENTRICLE ENLARGEMENT	2	2
RIGHT BUNDLE BRANCH BLOCK	2	2
NON PROGRESSION OF R WAVE IN VI-V3	26	26
PERSISTANT OF S WAVE IN V5-V6	18	18
NORMAL FINDINGS	26	26

In this study, 26 patients, i.e. 26% of patients have normal findings. 30 cases (30%) have sinus tachycardia. Non progression R wave in V1-V3 accounts for 26% i.e. 26 cases. Persistent of S wave in V5-V5 accounts for 18%, Low voltage complex account for 24%, Lead 1 sign is just 4% of cases.

ECG FINDINGS

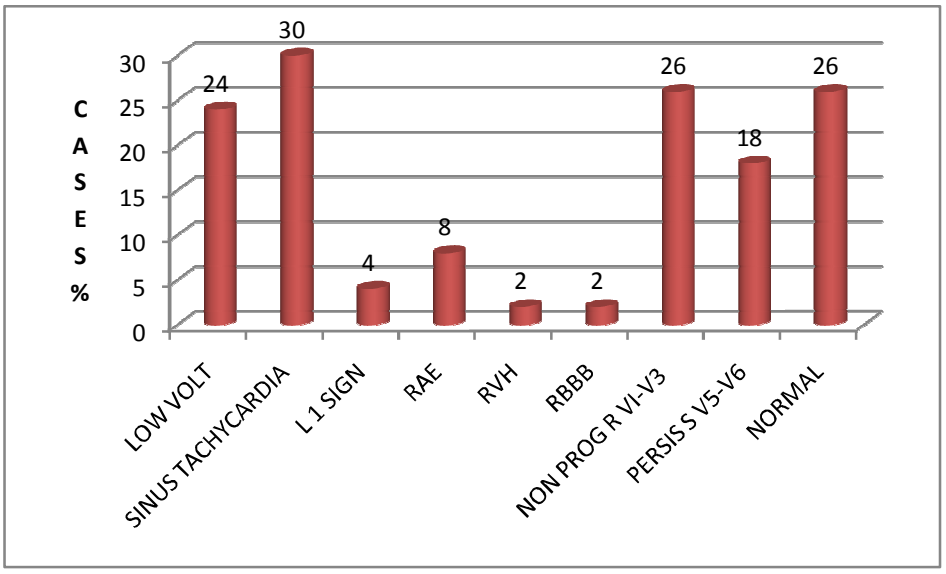


Table-18: HEMOGLOBIN VALUES IN STUDIED POPULATION

Hb in g%	male	%	female	%	total	%
<10	14	15	2	25	16	16
10 to11.9	36	39	6	75	42	42
12 to 14.9	42	46	0	0	42	42
>15	0	0	0	0	0	0
TOTAL	92	100	8	100	100	100

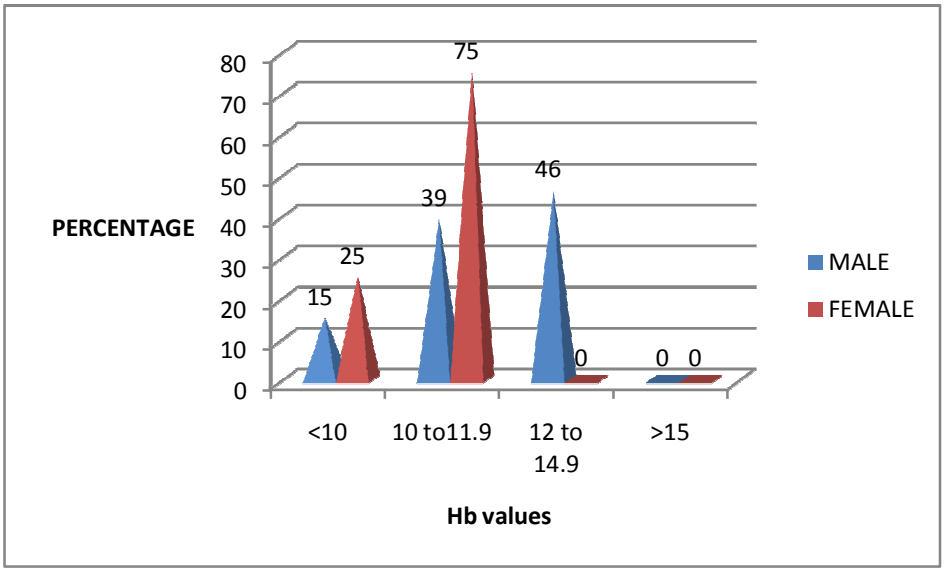


Table -19: HEMATOCRIT VALUES IN STUDIED PATIENTS

HEMATOCRIT VALUES IN %	NO OF PATIENTS	%
<35	60	60
36TO 39	26	26
40 TO 45	14	14
>45	0	0
TOTAL	100	100

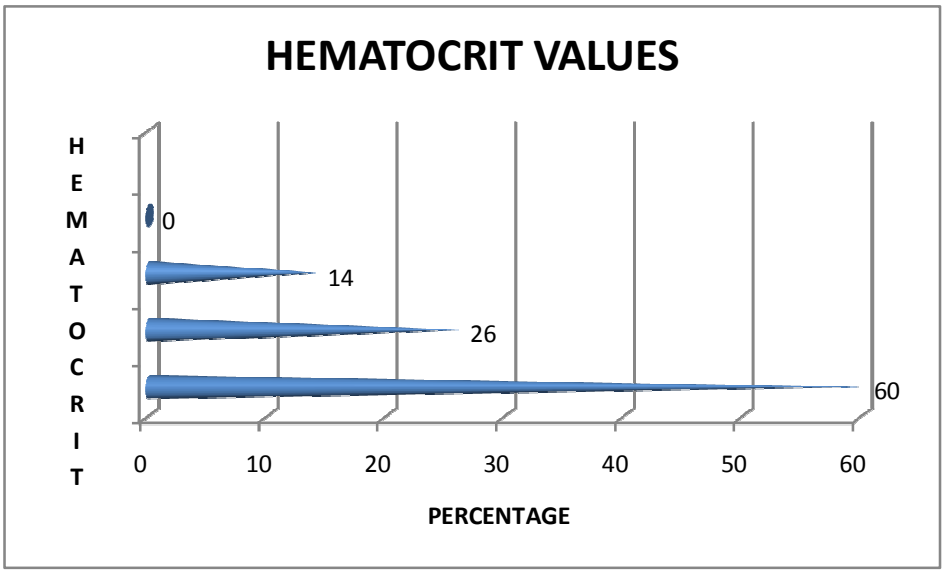
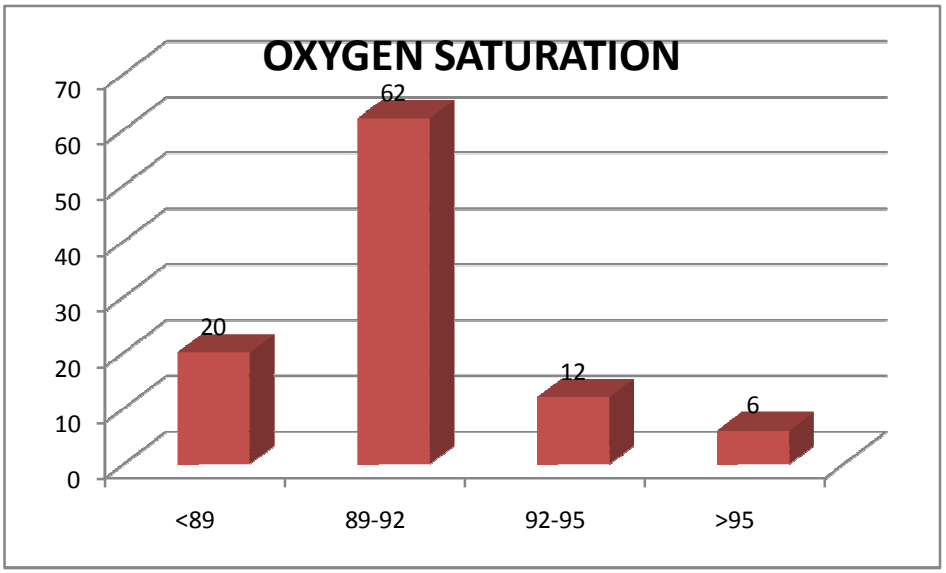


Table-20: PULSE OXIMETRY

SaO2	No of patients	Percentage
<89	20	20
89-92	62	62
92-95	12	12
>95	6	6

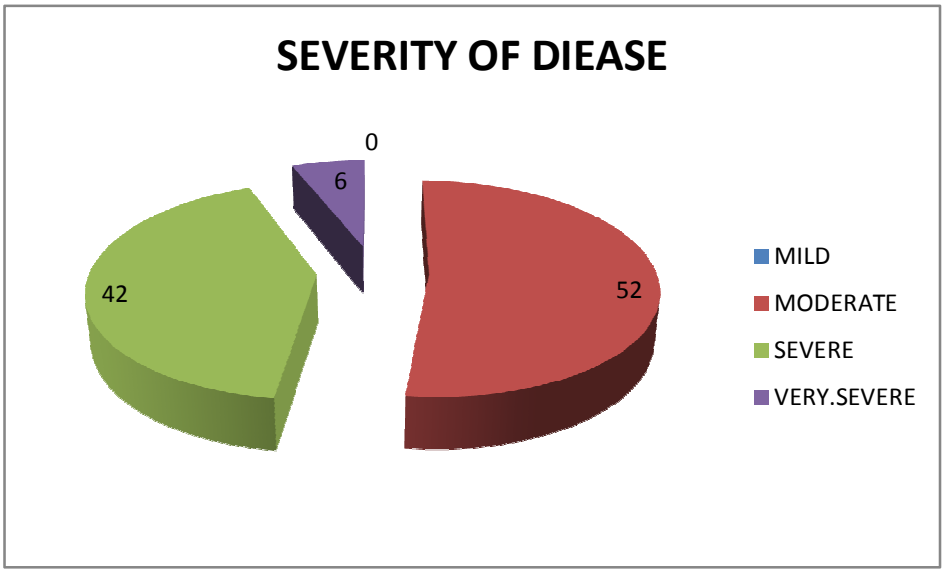


SPIROMETRIC VALUES

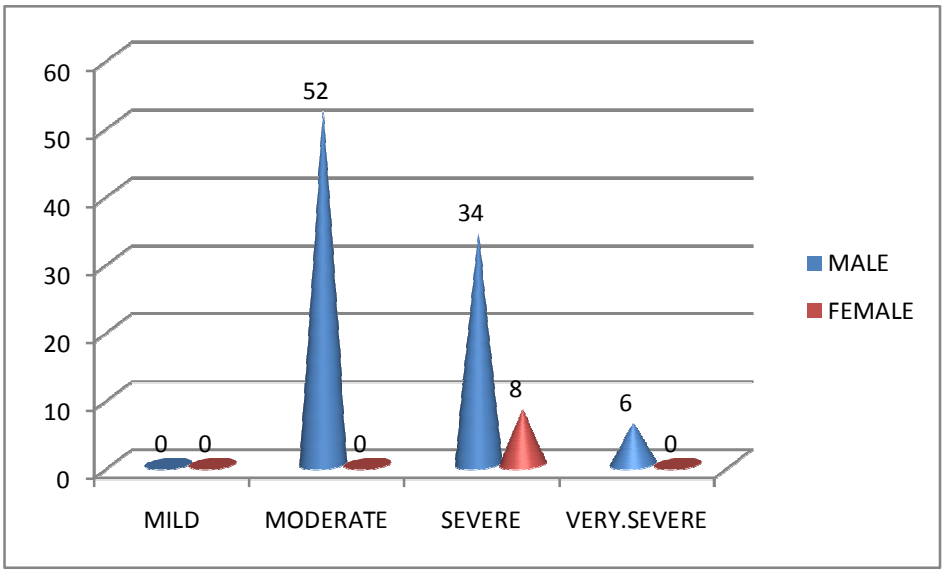
FEV1 and FEV1/FVC are considered as indices of pulmonary function in chronic obstructive pulmonary disease. FEV1 - reflects the degree of airway obstruction. FVC - Reflect the change in vital capacity. FEV1 / FVC< 70 shows obstructive lung disease.

Table-21: SEVERITY OF DISEASE IN STUDIED POPULATION

SEVERITY	MALE	%	FEMALE	%	TOTAL	%
MILD	0	0	0	0	0	0
MODERATE	52	56.5	0	0	52	52
SEVERE	34	37	8	100	42	42
VERY.SEVERE	6	6.5	0	0	6	6



BAR CHART SHOWING SEVERITY OF DISEASE



DISCUSSION

DISCUSSION

Age

Most of male patients were between 61-70 years of age constituting 50% and the Minimum number cases were in the age group of 81-90 years being 0%.

Among females, number of cases in the age group of 51-60 years constituted 50% and in the age group of 71-80 and 81-90 were one case each constituting of 25% each respectively.

Both sexes put together the maximum cases were in age group of 61-70 years constitute 46 % and minimum in the age group of 81-90 years constituting only 2%.

STUDY	41- 50	51-60	61-70
Sao paulo (Brazil)	9%	16%	25%
Santiago (Chile)	8%	14%	30%
Mexico city (Mexico)	3%	6%	18%
Montevideo (Uruguay)	5%	14%	32%
OUR STUDY (TRICHY, INDIA)	**8%**	**26%**	**46%**

With comparison of above studies, our study has highest percentage of COPD cases in 61-70 year age group.

SEX

92 percent of cases were male patients, females were 8 cases and 8 percent

STUDY	POPULATION	MALE, FEMALE RATIO
Radha (1977)	New Delhi	1.8
Thiruvengadam (1977)	Madras	1.6
Viswanathan (1966)	Patna	1.6
OUR STUDY	**TRICHY**	**11.5**

While comparison with, Our study has highest percentage of COPD cases in males

RURAL URBAN DISTRIBUTION

Our study shows 38 cases in rural area, 62 cases in urban population with 38% 1nd 62% respectively.

STUDY	POPULATION	RURAL, URBAN RATIO
Viswanathan (1977)	Delhi	0.67
Malik (1986)	North India	2.6
Jindal (1993)	North India	1.77
OUR STUDY	**TRICHY**	**0.61**

While comparing above studies, our study has lowest rural urban population ratio.

OCCUPATION

In this study group majority of the patients were non - agricultural coolies accounting to 54%. The remaining were businessmen and agriculture 20% each. House wives were accounting for 8%.

SMOKING

In our study out of 92 male patients 86 were smokers. In females 8 out 8 were non-smokers. Smoker, non smoker ratio is 6.1, it is much higher than other studies

STUDY	POPULATION	SMOKER, NON-SMOKER RATIO
Wig (1964)	Delhi	2.0
Sikand (1966)	Delhi	2.5
Ray (1995)	South India	1.6
OUR STUDY	**TRICHY**	**6.1**

PRESENTING SYMPTOMS

In the present study, cough and dyspnoea were present in all the cases and expectoration was present in 96% of cases, wheezing was present in 62% and fever was present only in 12% cases. It correlates with most of similar studies.

PHYSICAL SIGNS

In the present study, most of patients presented with obesity, short neck and Nicotine stain in lips, and very few had cyanosis. Some of patients were clinically pallor.

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RESPIRATORY SIGNS

In the present study majority of patients had crepitations and rhonchi and few patients had barrel shaped chest.

CHEST X-RAY

In the present study x-ray showed features of emphysema in 24 cases, features of chronic bronchitis in 20 cases & both emphysematous, bronchitis changes in 32 cases. 22 cases (22%) had no findings in x-ray chest.

ECG FINDINGS

In this study, 26 patients, i.e. 26% of patients have normal findings. 30 cases(30%) have sinus tachycardia. Non progression R wave in V1-V3 accounts for 26% i.e 26 cases. Lead 1 sign is just 4% of cases. Persistent of S wave in V5-V5 accounts for 18%.

So, general examination, complete systemic examination, chest x-ray and ECG need not to have findings in COPD patients.

HEMOGLOBIN

Most of the patients have below normal or near normal values. No case has the polycythemic range of Hemoglobin values. This may be due to malnutrition.

HEMATOCRIT

In this study 86% patients have less than 40% Hematocrit.

No single patient has value of polycythemia range. This may be due to other systemic causes.

In the study by Claudia Cote, MD, Marya Zilberberg, MD et al polycythemia was present in 5% of patients. In another study by C. Cote, S. H. Mody et al, polycythemia was present in 6% of cases.

PULSE OXIMETRY

In this study 20 cases (20 percent) have oxygen saturation less than 89%. Most of patients have 89-92% oxygen saturation. 6 patients have more than 95% SaO2.

SPIROMETRY

52% of patients were fall on moderate COPD, 42% were in severe COPD range. Only 6% of patients were in very severe range of COPD. Nobody was in mild COPD. This may be due to the study was conducted in inpatients who were admitted symptomatic.

CONCLUSION

CONCLUSIONS

- Majority of the patients were in the age group of 61-70 years. COPD was seen predominantly in male patients and majorities were smokers who have more than 20 smoke pack years.
- Because of urbanization, prevalence of COPD increasing in urban.
- In the majority of patients the duration of illness was 2-5 years. Cough and breathlessness were present in all patients.
- Wheeze is not the predominant symptoms in routine COPD patients.
- Diminished chest movements, crepitations, rhonchi, were present in majority of patients.
- As the number of cigarettes/day and duration increases the severity of the disease also increases in the studied population.
- In females exposure to smoke due to burnt fuel is the risk factor.
- In the study, about 40 % of cases were in stage III disease.
- As the severity & duration of the disease increases they are more prone to develop hypoxia and polycythemia as a complication. In our study 8 patients had hypoxia, as assessed by pulse oximetry, but no patient was in polycythemic range of hemoglobin or hematocrit values, this may be due to malnutrition.
- Clinical examination, ECG and chest X-ray may be normal in COPD patients. So diagnosis and severity should be based on Pulmonary Function Test.

- Pulse Oximetry is the best bedside tool to assess the hypoxemia in COPD patients and very useful to oxygen therapy.
- Spirometry is the definitive and gold standard investigation for diagnosis of chronic obstructive pulmonary disease, assess the severity of disease, reversibility and follow up.

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PROFORMA

PROFORMA

'An analysis of ECG, chest x-ray, pulmonary function tests, Pulse oximetry and Haematocrit abnormalities in chronic obstructive pulmonary disease patients'

I. Name

2. Age& Sex

3. IP NO

4. Occupation

5. Address

6. **Presenting complaints**

7. **History of present illness**

a. *Cough*-Duration/Dry or Productive If productive colour/odor/Quantity/Type Diurnal / seasonal / postural variation

b.Breathlessness- Duration/ Relation to exertion /variation/ Precipitating actors/PND,Orthopnoea

c. Wheezing Duration/Precipitating factors/Duration of attacks

d. Chest pain-Duration/Onset/Location/Nature/Radiation/Aggravating& Relieving factors

e. Hemoptysis-Duration/Quantity

f. Fever-Duration/Degree/Type/Association

8. **Past history** - Similar complaints / tuberculosis / asthma/ allergy / epilepsy / cardiac illness / diabetes mellitus/ hypertension

9. **Personal history**-Tobacco chewing/ Tobacco smoking-Pack years/Occupational exposure - Dust / fumes / smoke / chemicals

10. **Family history**-Tuberculosis/Bronchial asthma/Allergy like utricaria

Rhinitis / eczema

11. **General Examination**-Comfortable /dyspnoeic/Built/Nourishment/ Pallor/Cyanosis/Icterus/Clubbing/Oedema/Lymphadenopathy

12. **Vitals**-Pulse/BP/Respiration Rate/Pursed lip breathing

13. **Respiratory system**

Inspection-Shape of the chest/deformity/tracheal position/apex& other pulsations/ veins/Chest movement

Palpation-trachealposition/apex/chest movement/Vocal fremitus tenderness/Chest measurements

Percussion-note/Liver dullness

Auscultation-Breath sounds/Rhonchi/Crepitations/Pleural rub Vocal resonance

14. **Other systems**

15. **Investigations**

a) Hemoglobin/Red cell count/Differential count/ESR/ Haematocrit/ Random blood sugar/Urea/Creatinine

b) chest x-ray/ECG

c) Pulse oximetry- oxygen saturation/Pulmonary function tests FEV1, FVC, and FEV1/FVC after bronchodilation

16.**Diagnosis**

Treatment: Comments

MASTER CHART

Sl No	NAME	AGE/ SEX	OCU	DURA	SYMPTOMS	PACK YEARS	GE	PULSE	RR	CHEST	HB	RBC	HCT	ECG	CXR	FEV1 Pre	FEV1 Test	%	FVC Pre	FVC Test	%	FEV1/ FVC	SaO2
1	NATARAJ	60/M	C	2	Co,E,Dy,F	32	N	108	26	CMI,BSN,R+,Cr+	8.8	2.9	30	N	B,IBVM	1.87	1.22	65	2.31	1.91	83	64	90
2	JOSEPH	65/M	C	3	Co, E, Dy	25	Cl	82	20	BC, CMR,BSR, Cr+	12	4.0	34	L1,NPR, SV6	B+E,IBVM, HTL	1.76	1.08	61	2.2	1.62	72	67	92
3	MAHALINGAM	67/M	C	2	Co, E, Dy, W	20	N	92	18	CMN, BSN, Cr+	11.8	3.8	34	N	B,IBVM	1.69	1.12	66	2.13	1.59	74	69	93
4	VAIYAPURI	76/M	C	8	Co,E,Dy,W	24	N	98	24	CMI,BSN, R+, Cr+	12.4	4.0	32	NPR, SV6	B, IBVM,	1.46	0.74	51	1.89	1.27	67	58	90
5	PALANISAMY	54/M	B	6	Co,E,Dy,W	28	Cy	104	24	CMN, BSN, R+	11.6	3.8	30	ST	B, IBVM	2.12	1.10	52	2.42	1.58	65	69	89
6	SHANMUGAM	80/M	C	6	Co, E Dy	44	Pa	78	18	BC,CMR, BSR	10.8	3.2	30	L	E, HTL	1.42	0.84	59	1.78	1.24	70	68	94
7	SUSAIMANICKAM	57/M	B	6	Co,E,Dy,W	30	N	104	28	CMN,BSN, R+, C+	12.8	3.6	36	ST	B, IBVM	1.97	0.91	46	2.31	1.62	70	56	90
8	ALAGARSAMY	65/M	C	3	Co,E Dy, W	40	Cy	98	20	BC, CMR, BSR	11.4	4.2	34	L	E, HTL	2.47	1.14	46	3.05	1.84	60	61	95
9	GANESAN	58/M	C	3	Co, E, Dy	32	Pa	68	18	BC, CMN, BSN, Cr+	12.0	3.4	36	L	B+E,IBVM, HTL	2.43	1.42	58	3.0	2.15	72	66	98
10	SADIQ BASHA	60/M	A	7	Co, Dy	35	N	98	20	BC, CMR, BSR, R+	13.2	3.8	36	ST	E, HTL	2.08	1.10	53	2.42	1.78	74	61	92
11	MYLSAMY	70/M	B	20	Co,E,Dy,W	45	Pa	82	22	BC,CMN, BSN, Cr+	10.0	3.0	24	L	E, HTL	1.61	0.78	48	2.05	1.50	73	52	86
12	KRISHNAN	60/M	B	3	Co, E, Dy	30	Pa	102	24	BC, CMR, BSR, Cr+	9.2	3.5	28	L	E,HTL	2.24	0.85	37	2.58	1.53	60	56	88
13	VARADHARAJU	62/M	B	5	Co, E, Dy	-	Cl	88	18	CMN, BSN	10.2	3.4	32	N	N	1.89	1.04	56	2.31	1.72	74	60	96
14	GANAPATHY	72/M	A	11	Co, Dy	15	N	84	16	BC,CMN, BSN	11.8	3.0	34	L	E, HTL	1.84	1.02	55	2.24	1.62	72	63	92
15	MANOHARAN	69/M	C	10	Co,E,Dy,W	40	N	92	20	BC, CMR, BSR, R+	11.0	4.0	33	NPR, SV6	E, HTL	1.81	1.13	62	2.25	1.69	75	66	90
16	KESAVAN	54/M	A	6	Co,E,Dy,W	18	Pa	102	24	CMR, BSR, R+	10.4	3.1	28	N	B, IBVM	2.26	1.14	49	2.38	1.72	74	66	90
17	CHINNU	66/M	A	12	Co, E , Dy	30	Pa	98	24	CMN, BSN, Cr+	8.9	2.9	22	ST	B, IBVM	1.76	1.26	71	2.18	1.82	83	69	96
18	DURISAMY	70/M	C	7	Co, Dy	35	N	92	20	BC, CMR, BSN	12.4	4.2	36	NPR	E, HTL	2.21	1.08	49	2.65	2.21	83	49	94
19	ARUMUGAM	63/M	C	4	Co,E, Dy, W	22	N	94	20	BC,CMR, BSN	11.6	4.2	36	L1, NPR	E,HTL	2.26	0.88	39	2.60	1.72	66	51	90
20	RAHAMATHUNISHA	53/F	D	7	Co, Dy, W	-	Pa	104	24	CMR,BSN, R+,Cr+	11.2	3.8	37	ST, NPR	B+E, IBVM	2.14	1.06	49	2.68	2.33	87	45	90
21	NAGARAJ	78/M	C	4	Co, E, Dy,F	28	N	98	26	BC,CMN,BSN, R+,Cr+	13.6	4.4	42	N	N	1.46	0.90	62	1.89	1.44	76	63	92
22	KARUPPUSAMY	75/M	B	5	Co,E,Dy,W	45	N	92	24	CMN,BSN, R+,Cr+	12.4	4.2	38	ST	B, IBVM	1.89	1.29	68	2.56	1.88	73	69	90
23	RASATHI	84/F	D	14	Co,E,Dy,W,F	-	Pa	102	26	CMR,BSR, R+,Cr+	8.4	2.4	30	NPR SV6,ST	B. IBVM	2.60	0.86	33	3.12	1.76	57	49	88
24	MANIMUTHU	63/M	C	5	Co,E,Dy,F	30	Cl	98	26	CMN,BSN R+	12.8	3.8	36	N	N	2.24	1.54	69	2.74	2.28	83	67	91

OCU- occupation, DURA- duration of illness, M-male , F – Female , A- agriculture, B- Business, C- non agricultural coolie, D- house wife, Co- cough, E- expectoration, Dy- dyspnoea, W- wheeze, F- Fever,N- normal, I- increased, R- reduced Cl- clubbing, Cy- cyanosis, Pa- pallor, CM- chest movement, BS- Breath sounds, BC- barrel shaped chest, R-rhonchi, Cr- creps, ST- sinus tachycardia, RAE- right atrial enlargement, NPR- non progression of R-wave V1-V3, SV6- persistent S-wave in V5-V6, RVH- right ventricle enlargement, RBBB- right bundle branch block, B-bronchitis, E-emphysema, IBVM-increased broncho vascular markings, HTL-hyper translucency, FEV1-forced

Sl	NAME	AGE/ SEX	OCC	DU	SYMPTOMS	PACK YEARS	GE	PR	RR	CHEST	Hb	RBC	HCT	ECG	CXR	FEVI Pre	FEV1 Test	%	FVC pre	FVC Test	%	FEVI/ FVC	SaO2
25	MURUGESAN	75/M	A	7	Co,E,Dy,W	30	N	102	26	CMN,BSN,R+,Cr+	12.2	3.6	34	ST, NPR, SV6	B,IBVM	1.64	0.78	48	2.05	1.68	82	45	88
26	SELVARAJ	63/M	C	4	Co,E,Dy,F	28	Cl	98	24	CMN,BSN,R+,Cr+	11.8	3.8	32	ST	N	1.56	0.94	61	2.10	1.54	74	61	90
27	ABDUL MAJEETH	61/M	C	3	Co,E,Dy	35	N	106	26	BC,CMR,BSR, R+,Cr+	13.4	4.8	42	ST,NPR,SV6,L	E, HTL	1.94	1.12	58	2.33	1.89	81	59	88
28	VEERAMMAL	56/F	D	11	Co,E,Dy,W	-	N	104	24	CMN,BSN	11.2	3.4	32	N	N	2.32	1.14	49	2.82	2.13	76	54	90
29	CHINNAPPAN	69/M	G	12	Co,E,Dy,W	-	Cl	84	20	CMN,BSN, Cr+	14.2	4.6	40	N	N	1.76	1.15	65	2.20	1.68	76	69	90
30	VENKATESAN	48/M	C	8	Co,E,Dy,W	20	Pa	112	26	CMR,BSN,R+,Cr+	9.6	3.0	28	L	B+E IBVM,HTL	2.62	0.84	32	3.10	1.44	47	58	88
31	DURAIRAJ	73/M	C	10	Co,E,Dy,W	42	Cl	104	26	CMN,BSN,Cr+	13.2	4.4	40	ST	B, IBVM	1.54	1.03	67	1.97	1.66	84	62	92
32	RAJARAJAN	53/M	A	8	Co,E,Dy	25	N	96	24	CMN,BSN	12.4	3.8	36	N	N	2.52	1.48	59	3.34	2.70	81	55	90
33	MARIAPPAN	62/M	B	6	Co,E,Dy,W	25	N	98	22	CMR,BSR, R+,Cr	11.8	3.6	34	N	B, IBVM	1.81	0.95	52	2.25	1.46	65	65	92
34	SHAJAHAN	60/M	A	5	Co,E,Dy,W	32	Cy	114	30	CMR,BSN,R+,Cr+	14.8	5.2	44	ST,RAE,L	B+E IBVM,HTL	1.87	0.65	35	2.31	1.40	61	46	86
35	VADIVEL	43/M	C	3	Co,E,D,W	18	N	104	26	CMN, BSN, Cr+	12.6	3.6	34	ST	B, IBVM	2.65	0.68	26	3.11	1.28	41	53	90
36	ANNAMALAI	67/M	C	6	Co,E, Dy	30	Cl	110	28	CMN,BSN,R+,Cr+	14.2	3.8	42	ST,	N	1.69	0.96	57	2.13	1.42	67	68	91
37	NALLATHAMBI	62/M	C	11	Co,E,Dy,W	32	N	98	22	CMN,BSN,R+,Cr+	9.8	3.4	32	N	N	1.82	1.15	63	2.28	1.72	75	67	90
38	PAPPATHI	72/F	D	15	Co,E,Dy,W	-	N	106	24	BC,CMR, BSN, R+, Cr+	11.5	4.2	38	ST, L	E, HTL	2.72	1.03	38	3.27	1.96	60	53	91
39	SENTHAMARAI	58/M	C	4	Co,E,Dy,W	25	N	96	22	CMN,BSN, R+	11.2	3.6	34	N	B, IBVM	1.61	0.69	43	2.05	1.32	64	52	90
40	NATESEN	41/M	B	3	Co,E,Dy	22	N	98	24	CMN,BSN, Cr+	12.8	3.6	36	N	B, IBVM	1.96	0.94	48	2.62	1.82	70	52	92
41	ELUMALAI	70/M	C	12	Co,E,Dy,W	30	Pa	102	26	BC,CMR, BSN, R+	10.6	2.9	30	ST,NPR,	E, HTL	1.61	0.74	46	2.08	1.32	63	56	90
42	PANDIAN	52/M	A	2	Co,E,Dy,W	36	Cl	94	20	BC,CMR, BSR,R+.Cr+	11.4	3.4	32	RAE,RVH, NPR, SV6	B+E,IBVM,HTL	2.32	0.64	27	2.77	1.76	64	36	88
43	MANAMUNI	65/M	A	5	Co,E,Dy,	42	Cy	114	30	BC,CMR, BSR,R+,Cr+	14.8	5.2	42	ST	B, IBVM	2.07	0.66	32	2.51	1.32	53	50	86
44	SAMPATH	64/M	C	5	Co,E,Dy,W	18	Pa	98	24	CMN, BSN,	10.6	3.5	32	N	N	1.90	1.24	65	2.33	1.82	78	68	94
45	PERIASAMY	70/M	C	7	Co,E,Dy,W,F	35	N	97	26	BC,CMR, BSN, R+	13.2	3.8	38	L,NPR, SV6	E, HTL	2.64	0.94	36	3.12	2.42	78	39	88
46	AROKIARAJ	61/M	C	18	Co,E,Dy,W	38	Cl,	93	28	CMR,BSR,R+,Cr+	12.0	3.2	34	ST,L	B+E,IBVM,HTL	1.89	0.65	34	2.33	1.14	49	57	90
47	SUNDARAJAN	52/M	C	3	Co,E,Dy,W	25	Cy	98	30	BC,CMR,BSR, R+,Cr+	13	4.4	38	RAE,NPR,SV6	B+E,IBVM HTL	2.17	1.03	47	2.62	1.52	58	68	91
48	ANBALAGAN	70/M	B	7	Co,E,Dy,W	38	N	88	22	BC,CMR,BSN	11.8	3.6	32	RAE	E, HTL	1.98	0.61	29	2.35	1.49	63	41	92
49	JOHN	41/M	B	4	Co,E, Dy,W	-	N	94	20	BC,CMN, BSN	9.6	3.2	28	L	E, HTL	2.37	1.42	60	2.83	2.12	75	67	94
50	THANGARAJ	64/M	A	9	Co,E,Dy	42	N	86	18	CMN,BSN,R+,Cr+	12.4	3.5	34	RBBB	B+E,IBVM,HTL	2.87	1.68	59	3.56	2.92	82	58	92

Sl	NAME	AGE/ SEX	OCC	DURA	SYMPTOMS	PACK YEARS	GE	PR	RR	CHEST	HB	RBC	HCT	ECG	CXR	FEV1 Pre	FEV1 Test	%	FVC Pre	FVC Test	%	FEV1/ FVC	SaO2
51	SOLAIMUTHU	64/M	C	6	Co,E,Dy	30	Cy	110	28	CMN,BSN,R+,Cr+	14.2	3.8	42	ST	N	1.69	0.96	57	2.13	1.42	67	68	91
52	MANICKAM	74/M	C	11	Co,E,Dy,W	32	N	98	22	CMN,BSN,R+,Cr+	9.8	3.2	32	N	N	1.82	1.15	63	2.28	1.72	75	67	90
53	SINGARAM	68/M	C	4	Co,E,Dy,W	-	N	106	24	BC,CMR,BSN,R+,Cr+	11.8	3.6	34	N	B,IBVM	1.61	0.69	43	2.05	1.32	64	52	90
54	BALAMMAL	71/F	D	15	Co,E,Dy,W	25	N	96	22	CMN,BSN,R+	11.5	4.2	38	ST,L	E,HTL	2.70	1.01	38	3.27	1.96	60	53	91
55	THAMBIRAJ	42/M	C	3	Co,E,Dy	22	N	92	24	CMN,BSN,Cr+	12.8	3.6	36	N	B,IBVM	1.96	0.94	48	2.62	1.82	70	52	92
56	SOMASUNDARAM	55/M	A	6	Co,E,Dy,W	18	Pa	102	24	CMR,BSR,R+	10.4	3.1	28	N	B,IBVM	2.26	1.14	49	2.38	1.78	74	66	90
57	KARUPPAN	67/M	C	12	Co,E,Dy	30	N	92	20	BC,CMR,BSN	12.4	4.2	36	NPR	E,HTL	2.21	1.08	49	2.65	2.21	83	49	94
58	BALRAJ	70/M	C	7	Co,Dy	35	Pa	98	24	CMN,BSN,Cr+	8.9	2.9	22	ST	B,IBVM	1.76	1.26	71	2.18	1.82	83	69	96
59	AKILANDAM	64/M	A	4	Co,E,Dy,W	22	N	94	20	BC,CMR,BSN	11.6	4.2	36	L1,NPR	E,HTL	2.26	0.88	39	2.60	1.72	66	51	90
60	NAZARATH BEEVI	56/F	D	7	Co,Dy,W	-	Pa	102	24	CMR,BSN,R+,Cr+	11.2	3.8	37	ST,NPR	B+E, IBVM	2.14	1.06	49	2.68	2.33	87	45	90
61	VELANKANNI	61/M	C	18	Co,E,Dy,W	38	Cl	93	28	CMR,BSR,R+,Cr+	12.0	3.2	34	ST,L	B+E,IBVM,HTL	1.89	0.65	34	2.33	1.14	49	57	90
62	MOHAN	52/M	C	3	Co,E,Dy,W	25	Cy	98	30	BC,CMR,BSR,R+,Cr+	13	4.8	38	RAE,NPR,SV6	B+E,IBVM,HTL	2.17	1.03	47	2.62	1.52	58	68	91
63	DHAMODARAN	70/M	B	7	Co,E,Dy,W	38	N	88	22	BC,CMR,BSN	11.8	3.6	32	RAE	E,HTL	1.98	0.61	29	2.35	1.49	63	41	92
64	RAJ	41/M	B	4	Co,E,Dy,W	-	N	94	20	BC,CMN,BSN	9.6	3.2	28	L	E,HTL	2.37	1.42	60	2.83	2.12	75	67	94
65	AMALRAJ	64/M	A	9	Co,E,Dy	42	N	86	18	CMN,BSN,R+,Cr+	12.4	3.5	34	RBBB	B+E,IBVN,HTL	2.87	1.68	59	3.56	2.92	82	58	92
66	CHINNASAMY	72/M	C	10	Co,E,Dy,W	42	Cl	104	26	CMN,BSN,Cr+	13.1	4.4	40	ST	B,IBVM	1.54	1.03	67	1.97	1.66	84	62	92
67	SETTU	53/M	A	8	Co,E,Dy	25	N	96	24	CMN,BSN	12.4	3.8	39	N	N	2.52	1.48	59	3.34	2.70	81	55	90
68	SRIDHAR	62/M	B	6	Co,Dy,W	25	N	98	22	CMR,BSR,R+,Cr+	11.8	3.6	34	N	B,IBVM	1.81	0.95	52	2.25	1.46	65	65	90
69	ALAVUDEEN	60/M	A	5	Co,E,Dy,W	32	Cy	114	30	CMR,BSN,R+,Cr+	14.8	5.2	44	ST,RAE,L	B+E,IBVN,HTL	1.87	0.65	39	2.31	1.40	61	45	86
70	VELLAIYAN	43/M	C	3	Co,E,Dy,W	18	N	104	26	CMN,BSN,Cr+	12.6	3.6	34	ST	B,IBVM	1.81	0.95	52	2.25	1.46	65	65	92
71	NARASIMMAN	60/M	C	2	Co,E,Dy,F	32	N	108	28	CMI,BSN,R+,Cr+	8.8	2.9	30	N	B,IBVM	1.87	1.22	65	2.51	1.91	83	64	90
72	ANTHONYSAMY	65/M	C	3	Co,E,Dy	25	Cl	82	20	BC,CMR,BSR,Cr+	12	4	34	L1,NPR,SV6	B+E,IBVM,HTL	1.76	1.08	61	2.2	1.62	72	67	92
73	ALAGESAN	67/M	C	2	Co,E,Dy,W	20	N	92	18	CMN,BSR,Cr+	11.8	3,8	34	N	B,IBVM	1.69	1.12	66	2.13	1.59	74	69	93
74	MARUTHAI	76/M	C	8	Co,E,Dy,W	28	N	98	24	CMI,BSN,R+,Cr+	12.4	4.0	32	NPR,SV6	B,IBVM	1.46	0.74	51	1.89	1.27	67	58	90
75	PALANIVEL	54/M	B	6	Co,E,Dy,W	28	Cy	104	24	CMN,BSN,R+	11.6	3.8	30	ST	B,IBVM	2.12	1.10	52	2.42	1.58	65	69	89

Sl	NAME	AGE/ SEX	OCC	DURA	SYMPTOMS	PACK YEARS	GE	PR	RR	CHEST	HB	RBC	HCT	ECG	CXR	FEV1 Pre	FEV1 Test	%	FVC Pre	FVC Test	%	FEV1/ FVC	SaO2
76	SUBBURAJ	76/M	C	4	Co,E,Dy,W	28	N	98	26	BC,CMN,BSN,R+,Cr+	13.6	4.4	42	N	N	1.46	0.90	62	1.89	1.44	76	63	92
77	CHIDAMBARAM	72/M	B	5	Co,E,Dy,F	45	N	92	24	CMN,BSN,R+,Cr+	12.4	4.2	38	ST	B,IBVM	1.89	1.29	68	2.56	1.88	73	69	90
78	MARIAMMAL	82/F	D	14	Co,E,Dy,W,F	-	Pa	102	26	CMR,BSR,R+,Cr+	8.4	2.4	30	NPR,SV6,ST	B,IBVM	2.60	0.86	33	3.12	1.76	57	49	88
79	MANIMARAN	64/M	C	5	Co,E,Dy,F	30	Cl	98	26	CMN,BSN,R+	12.8	3.8	36	N	N	2.24	1.54	69	2.74	2.28	83	67	91
80	SERAN	77/M	A	7	Co,E,Dy,W	30	N	102	26	CMN,BSN,R+,Cr+	12.2	3.6	34	ST,NPR,SV6	B,IBVM	1.64	0.78	48	2.05	1.68	82	45	88
81	KANNAN	69/M	B	20	Co,E,Dy,W	45	Pa	82	22	BC,CMN,BSN,Cr+	10	3	24	L	E,HTL	1.61	0.78	48	2.05	1.5	70	52	86
82	BASKARAN	58/M	B	3	Co,E,Dy	30	Pa	102	24	BC,CMR,BSR,Cr+	9.2	3.5	28	L	E,HTL	2.24	0.85	37	2.58	1.53	60	56	88
83	ANADHAN	63/M	B	5	Co,E,Dy	-	Cl	88	18	CMN,BSR	10.3	3.4	32	N	N	1.89	1.04	56	2.31	1.72	74	60	96
84	BABU	74/M	A	11	Co,Dy	15	N	84	16	BC,CMN,BSR	11.8	3	34	L	E,HTL	1.84	1.02	55	2.24	1.62	72	63	92
85	SENTHIL	68/M	C	10	Co,E,Dy,W	40	N	92	20	BC,CMR,BSR,R+	11	4	33	NPR,SV6	E,HTL	1.81	1.13	62	2.25	1.69	75	66	90
86	RAMANATHAN	70/M	C	12	Co,E,Dy,W	30	Pa	102	26	BC,CMR,BSN,R+	10.6	2.9	30	ST,NPR	E,HTL,	1.61	0.74	46	2.08	1.92	63	56	90
87	SUNDARAJAN	55/M	A	2	Co,E,Dy,W	36	Cl	94	20	BC,CMR,BSR,R+,Cr+	11.4	3.4	32	RAE,RVH,NPR,SV6	B+E,IBVM,HTL	1.32	0.64	27	2.77	1.72	64	36	88
88	RAFIQ	67/M	A	5	Co,E,Dy	42	Cy	114	30	BC,CMR,BSR,R+,Cr+	14.8	5.2	42	ST	B,IBVM	2.09	0.66	32	2.51	1.32	53	50	86
89	KANDASAMY	61/M	C	5	Co,E,Dy,W	18	Pa	98	24	CMN,BSN	10.6	3.5	32	N	N	1.90	1.24	65	2.33	1.82	78	68	94
90	PARAMESWARAN	68/M	C	7	Co,E,Dy,W,F	35	N	97	26	BC,CMR,BSN,R+	13.2	3.8	38	L,NPR,SV6	E,HTL	2.64	0.94	36	2.12	2.42	78	39	88
91	SIVAKUMAR	78/M	C	6	Co,E,Dy	44	Pa	78	18	BC,CMR,BSR	10.8	3.2	30	L	E,HTL	1.42	0.84	59	1.78	1.24	70	68	94
92	KANAGARAJ	58/M	B	8	Co,E,Dy,W	30	N	104	25	CMN,BSN,R+,Cr+	12.8	3.6	36	ST	B,IBVM	1.92	0.91	46	2.31	1.62	70	56	90
93	ARIVAZHAGAN	62/M	C	3	Co,E,Dy,W	40	Cy	98	20	BC,CMR,BSR	11.8	4.2	34	L	E,HTL	2.47	1.14	46	2.05	1.84	60	61	95
94	RAJAGOPAL	53/M	C	6	Co,E,Dy	32	Pa	68	18	BC,CMN,BSN,Cr+	12	3.4	36	L	B+E,IBVM,HTL	2.42	1.42	58	3.0	2.15	72	66	98
95	VISHNU	59/M	A	7	Co,E,Dy	35	N	98	20	BC,CMR,BSR,R+	13.2	3.8	36	ST	E,HTL	2.08	1.10	53	2.42	1.78	74	61	92
96	THULASIDAS	62/M	C	4	Co,E,Dy,F	28	Cl	98	24	CMN,BSN,R+,Cr+	11.8	3.8	32	ST	N	1.56	0.94	61	2.10	1.54	74	61	90
97	SRIRANGAN	65/M	C	3	Co,E,Dy	35	N	106	26	BC,CMR,BSR,R+,Cr+	13.4	4.8	42	ST,NPR,SV6,L	E,HTL	1.94	1.12	58	2.33	1.89	81	59	88
98	NARAYANAN	63/M	D	11	Co,E,Dy,W	-	Cl	84	20	CMN,BSN,Cr+	14.2	4.6	40	N	N	1.76	1.15	65	2.20	1.68	76	69	90
99	PONNAMMAL	58/F	C	12	Co,E,Dy,W	-	N	104	24	CMN,BSN	11.2	3.4	32	N	N	2.32	1.14	49	2.82	2.13	76	54	90
100	LINGUSAMY	46/M	C	8	Co,E,Dy,W	20	Pa	112	26	CMR,BSN,R+,Cr+	9.4	3.0	28	L	B+E,IBVM,HTL	2.62	0.84	32	3.10	1.44	47	58	88

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This is to certify that the project work titled " **An analysis of E.C.G.,chest X-ray, Pulmonary function tests, Pulse oximetry,hematocrit abnormalities in chronic obstructive pulmonary disease patients"**proposed by Dr.P.Sasikumar of K.A.PV.Govt.medical college, Tiruchy as part of fulfillment of M.D course in the subject of ……General Medicine…… for the year …2011-12…….. by The Tamilnadu Dr.MGR medical university has been cleared by the ethical committee.

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